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CLINICAL EXAMINATION OF THE NERVOUS SYSTEM

MONRAD-KROHN

FIFTH EDITION

For Review.

The Clinical Examination of the Nervous System. By G. H. MONRAD-KROHN. New York: Paul B. Hoeber. 5th ed., 1930. xvi + 222 pages. Price, $3.50.

Monrad-Krohn's excellent small book, now in its fifth edition, was first published in 1921. It at once became the foremost book of its kind, in English, and, for nearly ten years, has held the field in spite of considerable opposition. The style is clear, concise and easy to read and the subject is more completely covered in this book than any other of its size. In the new edition, one finds many references to recent American literature on neurological diagnosis. The author is to be congratulated on his keenness in following the latest additions to our knowledge of neurological examination.

N. E. J. M. 8/21/30

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THE CLINICAL EXAMINATION OF THE NERVOUS SYSTEM

BY

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FIFTH EDITION

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FOREWORD

ALL who have had experience of neurological work realize how essential it is to have a routine method of examination of the nervous system. By the employment of some definite method, time is saved, mistakes due to overlooking symptoms and signs are safeguarded against, the case becomes focussed in the mind, and any points which require further investigation are brought out clearly.

The student, house physician or general practitioner who is without such a method is placed at a great disadvantage when confronted with a case of nervous disease.

There can be no doubt that in the past the tendency to separate Neurology from Psychiatry has done much to hinder the progress of both these branches of medicine, and it is a welcome sign of the times to see that the trend of modern opinion is to bring them closer together.

All who intend to make their special study disorders of the nervous system, whether it be neurology or psychiatry, should have as a foundation a knowledge of neurology, psychology and psychiatry. It is impossible to divorce the physical from the psychical, or the psychical from the physical, and this is true of every branch of medicine.

Dr. Monrad-Krohn in his book upon "The Clinical

Examination of the Nervous System," describes a routine method of examination which he has adopted in his neurological clinic, and has given an outline of the clinical tests which he himself considers to be the most practical and useful.

He has shown his appreciation of the true needs of the neurologist and psychiatrist, and has not confined his book to the purely neurological aspect of nervous disease, but includes an outline of the examination of the mental state of the patient. He insists upon the importance of combining a knowledge of neurology with that of psychiatry, and emphasizes the fact that proficiency in neurological examination can only be obtained through practice, and that his book is intended for use in close connection with clinical work.

To the general practitioner it will afford an opportunity of making himself conversant with various tests, and practice will enable him to apply them to the investigation of many cases the true nature of which he would be unable to solve without such knowledge.

Dr. Monrad-Krohn is a Member of the Royal College of Physicians, London,* and has had a wide experience of the practical side of both neurology and psychiatry. Having studied neurology in England, he is well versed in the methods of the British School, and his book therefore is of special value as it is the work of a Norwegian who has studied the subject in other countries as well as here, and is thus able to present a scheme of examination of the nervous system based on a wide range of personal experience.

* Since 1927 a Fellow of this College.

The book is not a translation, but has been written in English by Dr. Monrad-Krohn himself. He is to be congratulated upon this, and if at times his mode of expression may strike the reader as unfamiliar, he always succeeds in making his meaning clear and emphasizing his point. The book is free from that vagueness of meaning which is not uncommon in many translations, and will, I feel sure, be welcomed not only by students and those engaged in general practice, but by all who are interested in the study of disorders of the nervous system.

T. GRAINGER STEWART.

LONDON, 1921.

PREFACE TO THE FIFTH EDITION

For the present edition I have again revised the book, and have made a number of minor additions and alterations. A short description of the methods of ventriculography and encephalography has been added.

In the chapter on repeated examinations I have added a description of the use of hypertonic solutions as means of reducing the intracranial tension and thereby rendering a dazed patient co-operative for a subsequent examination. In the choice and description of the various methods I have now as before chiefly been guided by experience gained in the daily work in my clinic, where I have had the advantage of the valuable assistance of Drs. Lossius, Forsberg, Schjött, Östrem, Ingebrigtsen, and Holst-Larsen.

I wish particularly to thank my senior assistant physician, Dr. Lossius, for his great help in correcting the proofs.

G. H. MONRAD-KROHN.

Oslo, *March* 1, 1930.

PREFACE TO THE FIRST EDITION

" Recueillir les faits et ne s'astreindre à les interpréter qu'ensuite est la condition indispensable pour arriver à la verité."—CLAUDE BERNARD.

IN neurology more than in any other branch of medicine the diagnosis is built up of a number of details found by clinical examination. It is the main object of the examination, then, to furnish *all* the details, to elicit *all* the functional disturbances that the case presents, and in order to do this the clinical examination has to be both systematic and complete.

Every budding neurologist invariably experiences a great deal of difficulty in establishing for himself a satisfactory routine method of examining, and it is here that the author intends to help the beginner by giving him an outline of the clinical tests in the order found most practical in the neurological clinic.

It is hoped, then, that the book will be of some use to the young neurologist, to the house physician in making his first complete neurological examinations, and also to the general practitioner in refreshing his memory. It is unnecessary to point out the practical importance of this systematic neurological examination. How often

does it not disclose a cerebral tumour, when the patient just came " to get some medicine for a headache "!

The clinical examination is here given in the form which the author utilizes in his clinical teaching, and he has had ample opportunity to ascertain that the students very soon learn to make a reliable examination following the instructions here given. Only those tests are given which the author has satisfied himself are practical and useful. The present book is in the main only an enlarged edition of a Norwegian book on the same subject, published in 1914.

But some additions have been made, including a somewhat more detailed account of the mental examination. It is recognized more and more that psychiatry and neurology are to a very high degree interdependent.

The neurological examination, without at least an honest attempt to investigate the patient's mental condition, must be regarded as incomplete. It is fully realized that the correct estimation of a patient's mental state often necessitates continued observation during many days and weeks. What this book proposes to give is only an outline of the first mental examination, which in the majority of cases will give the examiner a rough orientation as regards the patient's mentation.

On the other hand, it cannot be emphasized too strongly that the psychiatric examination is equally incomplete unless it is accompanied by a complete neurological examination; and the author therefore

ventures to hope that this little book may also be of some use to the medical officer of the mental hospital, who feels the need of supplementing his mental examination by a *complete* neurological examination.

Although this is not intended to be a book on the diagnosis of nervous diseases, a few diagnostic considerations have been introduced, mainly to illustrate the significance of various tests.

It must be emphasized that proficiency in the neurological examination can only be attained through practical work; this book is therefore intended for use in close connection with the clinic.

The author recommends his students the following procedure: First, complete the whole systematic examination; give the examination your whole attention without speculating about the diagnosis till the examination has been completed. Then write out a tabulated list of your findings; it is most convenient to arrange them in two columns corresponding to the two sides:

Right.	Left.

Next, try to arrive at a *focal diagnosis* based on your anatomical and physiological knowledge. Finally, consider the *nature* of the lesion, aided by your knowledge of general pathology.

Only in this way can the student be trained to arrive at a diagnosis in cases which are not typical and which

he does not find described in his textbook. Nothing is more injurious to the student's training than the usual way of proceeding, in which the student first reads a textbook, and then, without any logical reasoning, tries to remember what type of disease the case "reminds" him of, with the final conclusion that the case "looks like" this, that, or the other.

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THE CLINICAL EXAMINATION OF THE NERVOUS SYSTEM

ANAMNESIS.

THE way of ascertaining the different data regarding the patient's family, his previous life, and the history of the present illness, is the same as elsewhere in medicine. The patient should be allowed to tell his history in his own words; he should be listened to patiently, all suggestion being carefully avoided. To complete the history, a few leading questions may have to be asked, particularly about the following symptoms:

Headache (constant or intermittent, diffuse or localized; hemicrania).

Vomiting, with or without subjective feeling of nausea.

Vertigo.—Is it more pronounced in the dark than in light? "wash-basin symptom"? tendency to fall to one side? any subjective sensation of rotation?

Disturbances of Vision.—First and foremost *diplopia* (frequent in "epidemic" encephalitis, disseminated sclerosis, tubercular and syphilitic meningitis).

Pains and Paræsthesia (N.B.—Initial root pains in extramedullary tumours—"cushion sensation," "girdle sensation," in tabes, *e.g.*).

Disturbances of Consciousness (loss of consciousness, automatism, petit mal).

Convulsive Fits (tonic or clonic, general or localized, followed by localized paresis; Jacksonian fits).

Both in regard to disturbances of consciousness and convulsive fits one must ascertain if the patient has any recollection of what has happened. If not, total amnesia is said to exist for the attacks—a characteristic point in genuine epilepsy.* Also it should be ascertained if the patient has any kind of previous warning (aura). An exact description of this should be obtained.

Bladder Trouble.—Precipitate micturition (frequent in disseminated sclerosis); incontinence; retention; ischuria paradoxa (incontinence by overflow).

Constipation.—There is no doubt that many neurotic complaints are accentuated by constipation.

Disorders of Sleep: Insomnia—Hypersomnia (as detailed an account as possible).—In insomnia is it the process of going to sleep that is impeded by pains, by persevering thoughts or by anxiety? Or does the patient wake up with any peculiar sensation? How many hours does the patient sleep during a night and a day?

It is of great importance to ascertain whether the illness started *suddenly* (a certain day or a certain hour; this generally indicates a vascular "catastrophe"—viz., hæmorrhage, embolism, thrombosis) or *gradually* (*e.g.*, tumours, degenerative lesions), and if there have been *remissions* (as, *e.g.*, in disseminated sclerosis).

* In all such cases the *hyperventilation test* should be carried out (cp. p. 99). Also a complete urine examination *à la* Bisgaard (quantitative tests for NH_3 and total N, and the concentration of H ions) should be carried out with a view to deciding the presence of "dysregulatio ammoniaci." As these tests fall outside the scope of this book, the reader is referred to Bisgaard's article in *Comptes rendus de la Société de Biologie*, 1921

Inquiries about *syphilitic infection* should always be made, if not directly, then indirectly—abortions, still-births, rash, etc. Intemperance with regard to *alcohol* and *tobacco* should be noted, and the quantity consumed should be stated as accurately as possible.

STATUS PRÆSENS.

In every case a complete examination of the lungs (purulent affections of the lungs may lead to metastatic abscess of the brain), the heart (N.B.—cerebral embolism by valvular—particularly mitral—disease), and the abdominal organs is made in the ordinary way. Temperature, pulse, and respiration are also noted, and the urine must be tested. (N.B.—Important relations of nephritis to cerebral hæmorrhage, of diabetes to neuritis and neuralgia—"pseudo-tabes diabetica.") The blood should also be examined (pernicious anæmia may lead to "subacute combined sclerosis," leucæmia to compression of the cord and nerves, polycythæmia vera may produce a clinical picture resembling that of cerebral tumour). The blood-pressure should also be measured (increased blood-pressure predisposes for cerebral hæmorrhages). It is of little importance whether all this is done before or after the examination of the nervous system; but the patient must not be tired out, as the exact neurological examination of a weary patient is very difficult.

1. MENTAL STATE.

There is no hard-and-fast method for eliciting every mental symptom; only in a broad way the points to which the observer should direct his attention may be indicated.

To include a complete psychiatric examination in our routine neurological examination is unfortunately impossible for practical reasons. But the state of the patient in regard to the following should be ascertained:

(1) **Attention.**—Is it easily obtained and kept? or can it not be roused (*e.g.*, catatonic stupor)? or is it apt to wander (divertibility of attention in mania)? Particular tests for attention may also be employed—*Bourdon's test* consists in the patient's striking out certain letters in a page of a book—*e.g.*, every *r* he sees. If the result is timed one must always employ the same standard page and the same letter for purposes of comparison.

(2) **Perception.***—Does the patient understand what is said to him? Does he recognize objects shown him?

(3) **Orientation in Time and Space.**—Can the patient give the time, day, date and year? Can he tell where he is? Does he recognize his surroundings?

(4) **Memory—**

(α) *For Recent Events.*—What did he have for breakfast and lunch? How long has he been sitting in the waiting-room? What did he do yesterday, etc.?

* A *complete* examination of the patient's perception—*i.e.*, all his psychosensory functions—cannot possibly be included here. It will be dealt with later in the Appendix. Errors of perception (illusions and hallucinations) will also be mentioned later (*cf.* p. 10).

Tests with Figures and Stories.—Normal adults of average intelligence* are able to repeat six numbers forwards and five numbers backwards—*e.g.*, 4 5 3 1 9 6, 9 3 5 2 1 7; 4 8 7 6 3, 3 5 9 8 4. Give the patient an address—*e.g.*, 92, Fleet Street—and ask him after three to five minutes for it. This is not a simple test for memory; attention also enters into it.

Tell him a short story and let him repeat it.

(β) *For Remote Events.*—When was he born, particulars about his family, school, occupation, holidays and their meaning, Christmas, Easter, etc.

It is also important to test the patient's *memory for time relations* of various past events ("recordation")—*e.g.*, when the patient has given the date and year of his wedding correctly, one proceeds to ask him: "Do you remember any other events that took place in the year of your wedding? What was your position then? Where did you live? Who was Prime Minister then?" etc. (chiefly questions from his particular sphere of interests).

Does the patient fill in the gaps in his memory with fabrications (*paramnesia*—Korsakoff's syndrome)?

(5) **Reasoning Powers.**—What does the patient think of himself, of his family, of others? Conversation about his occupation, business, position; his views, political and others.

Helpful to get a more definite idea of the patient's reasoning powers are *Ziehen's tests:* Define the distinction between mistake and lie, dwarf and child, horse and ox, water and ice. *Finckl's tests:* Explain the meaning of proverbs, "Set a thief to catch a thief." "The early bird catches the worm."

* Viz., about 75 per cent. of all adults.

The Masselon test: The patient is asked to form a sentence containing certain words—*e.g.*, needle, button, thread—hunter, dog, gun, forest, rabbit—pen, ink, letter.

Ebbinghaus's test may also be found useful. The patient is asked to complete a sentence in which several words have been left out—*e.g.*: "I went out in the . . . and picked some . . . roses. I put them in a . . . on the . . . and their deep . . . colour appeared redder still against the snowy . . . of the tablecloth." "I got up in the . . ., and after washing and brushing my . . . went to . . ."

So-called *ethical tests:* "What would you do if you saw a man drop a five-pound note?" Such oral tests have limited value. The patient might quite well say what would be the right thing to do, yet he might possibly do the wrong thing (moral insanity).

The Binet and Simon system of tests: See Appendix, p. 151.

Has he any *delusions*?*—grandiose delusions—delusions of unworthiness, of sin, of impending harm, "nihilistic" delusions? (The patient believes that he has no heart, no stomach, or no lungs, etc.) Delusions of persecution—are people against him? Is there a plot against him (paranoia and dementia paranoides)?

* Delusions are *erroneous beliefs impervious to reason;* they may be so *absurd* that the delusional character is at once apparent (*e.g.*, a general paralytic's belief that he is Emperor of China), but may, on the other hand, often be so *plausible* that the patient's friends and relations have to be questioned very closely in order to ascertain the delusional character of the patient's statements. In the very early stages of paranoia this may sometimes be very difficult and requires great care.

Are the delusions fixed or changing? Is there *one* system of delusions?

As long as dementia has not supervened there will always be a certain systematization of delusions. It is a *normal* mental feature to bring one's ideas into harmony with one another. But *one* fixed system of delusions, arranged round a central "nucleus"—an original delusion, from which all the others are derived—is characteristic of *paranoia.*

Arithmetical Powers.—"You have 2s. 6d. in your pocket. You buy 10d. worth of fruit and 9d. worth of flowers; how much change have you left?" Simple additions and multiplications. Characteristic is the way in which general paralytics with grandiose delusions often, without hesitation, give enormous sums in answer to the simplest arithmetical question. "No stinting," as one of the author's patients remarked himself, stating that three fours equalled "five thousand pounds"!

(6) **Emotional State.**—*Emotional tone,* gay in mania, depressed in melancholia; *emotional outbursts* and *emotional control. Indifference* and *apathy* in dementia præcox and in frontal tumours. *Euphoria* in dementia paralytica. *Incontinence of emotional expressional movements* in "pseudo-bulbar paralysis" and many cases of disseminated sclerosis.

(7) **Volitional Functions.**—His will and power of endurance and perseverance. His career. His wishes and desires. (The psychomotor disturbances—aphasia, agraphia, and apraxia—will be dealt with later, in the Appendix.) Here any *obsessional actions* and *impulses* will be noted—*e.g.*, "washing mania," ob-

sessional impulses to touch certain things a certain number of times, to count (arithmo-mania), etc. These obsessions are closely related to the various forms of "tic," which is but the same phenomenon on a lower level of the central nervous system;* they are also closely related to the different forms of *phobia—e.g.*, claustrophobia, agoraphobia, etc. *Folie de doute*, an endless fight between impulses and counter-impulses, with no decisive victory for either.

The following volitional disturbances are frequently found in dementia præcox. *Negativism*, in which a suggestion immediately calls up an overwhelming counter-suggestion, with the result that the patient does just the opposite of what he is asked to do; sometimes this overwhelming counter-impulse is mobilized after the suggested movement has started. This results in the movement being stopped in the middle ("blocking of will"). When trying to shake hands with such a patient, the patient will either put his hand behind his back, or he will start putting his hand out, then stop the movement, and withdraw the hand without shaking hands.

It is a good plan to make a habit of shaking hands with the patient. Just as the dementia præcox patient has a peculiar way of shaking hands, so has also the melancholiac and the maniac. The maniacal handshake is from the shoulder (a movement of the whole arm); the melancholiac handshake is from the wrist, the rest of the arm being kept fixed in an attitude

* Tic may quite well be defined as a *motor obsession* or an *obsessional movement*.

resembling paralysis agitans. When dealing with a violent patient, it is a precaution worth taking to make sure of his right hand by means of a prolonged handshake.

In *automatic obedience* the patient, on the other hand, immediately follows any suggestion; in *echolalia* he repeats* everything that is said, or the last of every sentence he hears; in *echopraxia* the patient repeats* the observer's movements; in *catalepsy* a limb is kept in the position in which the observer puts it, as if the patient forgets it. There are various degrees of *catalepsy*. When not very marked, it is difficult to say whether it is present or not. Helpful in deciding this question is the following manner of proceeding: The patient's arm is passively lifted to the horizontal and left in this position;† the patient is then, after one or two seconds, asked to rise or to sit down or to put out his tongue. Normally, the patient will then allow his arm to sink down; if the arm still remains up, catalepsy is said to be present. Just as the patient sometimes repeats everything that is said or done before him (echolalia and echopraxia), he often repeats what he himself says and does‡ (*palilalia, stereotypy*). The verbal repetitions often consist of senseless words formed by the patient himself (neologism), and is then called *verbigeration*. The motor repetitions may consist of all sorts of weird antics (mannerisms).

* Repetition is the *cheapest* form of innervation. When volition is waning, repetition becomes more frequent.

† This should be done casually, as if by accident.

‡ See note * above.

Finally, *illusions* and *hallucinations** must be carefully investigated—aural ("voices"), visual ("visions"), olfactory, gustatory, tactile—often in a roundabout way. Often hallucinations will have become evident when delusions are ascertained, as delusions and hallucinations are frequently connected.

In visual hallucinations it should be ascertained if they are *hemianopic*. *Simple* hemianopic hallucinations (*e.g.*, flashes of light confined to the right or the left halves of the visual fields) point to a one-sided occipital irritation (most commonly a tumour of the occipital lobe). *Complex* hemianopic hallucinations (persons or animals moving about in homonymous halves of visual fields) point to an irritative lesion (most commonly tumour) of the temporal lobe.

The so-called *uncinate attacks* are of importance, pointing to an irritative lesion (tumour, scar after a trauma) in the vicinity of the uncinate lobe. These uncinate attacks consist in—

1. Olfactory or gustatory hallucinations (mostly of an unpleasant character—"an evil smell," "an evil taste").
2. A slightly dazed condition.
3. A feeling of going through some past experience again (often of unpleasant character).
4. Smacking movements of lips and jaw.

Also it must be ascertained whether the patient has any *insight*—viz., the understanding that he is mentally not quite normal.

Example of mental disturbances with insight: obsessional insanity.

Example of mental disturbances without insight: paranoia.

* An hallucination is a perception *without* any external stimulus causing it. An illusion is a perception arising by misinterpretation of a sensation.

When making notes of the mental condition of a patient, a special point should be made of recording the replies in the patient's own words as much as possible. No judgment about the patient should be recorded without substantiating it by actual facts from the examination.

The ordinary *association tests* may be useful; they are dependent on a number of different factors: attention, state of rest or fatigue, perception, emotional value of the associations concerned, and psychomotor functions; they must therefore be interpreted with great reserve. The patient is asked to answer with the first word that comes into his head, the observer mentioning in succession a number of words taken from a list, which anybody can make up for himself—*e.g.*, "wood, house, disease, small, town, guilt, mother, sweet, prison, wedding, death, water, colour, ship, moon, mountain, lake, fish, dog, table, dwarf, swim, stone, road, money, envy, justice, negro, law, stick, father, grass, children, meal, clouds, thief, face, fruit, book, fingers, murder, hour, review, music, comfort, anger, hesitation, kiss, mirror, dance, kill, think, sun, hate, sweetheart, prince, secret, good, horse, love, church, poison, king, plot, wine, happiness," etc.

The answers, as well as the reaction times, are noted (a fifth of a second stop-watch being used). The value of the association tests is best gleaned from the following description of Peterson's:*

* "Textbook of Nervous and Mental Diseases," Church and Peterson. Saunders. 1916.

"While the word-association method is of some use for the purpose for which it was first employed—viz., to estimate the intelligence of an individual—it is of even greater value in uncovering emotional complexes. The subconscious is vastly more important to us than the conscious, for in the subconscious lie all the elements that make up our personality, not only the treasury of all our individual experiences through the course of years, but all our ancestral trends, desires, tendencies, wills, ambitions, controls, inhibitions, fears — in fact, the latent spirit of the race of mankind.

Each man's vocabulary, be it the three hundred words of the sailor, or the fifteen thousand words of a Shakespeare, or the average fifteen hundred or two thousand words belonging to us, is related to all that subconscious material. A word has a magic power in it to summon from the vaults of memory all sorts of apparitions. Each word has an emotional value, some more than others, because all of our deepest experiences are associated with the words we know.

So when an apparently empty word is propounded to a patient, and he is asked to answer as quickly as possible with the first word that comes into his mind, we not only obtain an association from his memory storehouse, but we may strike some emotional complex which is indicated by a slow response to the test word or retarded reaction time.

This is the word-association method of Jung, a method that is employed for the discovery of secrets in the criminal, or painful and disease-producing emotional complexes in patients suffering from various psychogenic disorders. In making this test, just ordinary, everyday words are used, since these are the especial words related to an ordinary individual's experiences, and a fifth of a second stop-watch is used to measure the reaction time. An emotional complex is so apt to have many words associated with it that there is an inrush of many words to the stimulus word, and the mind pauses for a choice; hence the retarded reaction time. Having gone over the list once with a stop-watch, we go over the same list of words again to see how well the first associa-

tions are remembered. The inrush of words is responsible for faulty memory here, and where there are emotional complexes these reproductions are apt to be false, some new word being associated the second time.

Thus, a patient was given a series of unimportant words that had no significance whatever to the investigator, and among them the following had three or four times as long reaction time as the others, so that it was clear that an emotional complex lay behind them: Water—deep, 5 seconds; ship—sink, 3·4 seconds; lake—water, 4 seconds; swim—can swim, 3·8 seconds.

Psycho-analysis showed that the patient had recently been depressed, and had determined to commit suicide by drowning."

The above investigation of the different psychic faculties is in itself a psycho-analysis, inasmuch as every mental examination is a psycho-analysis. The particular form of "psycho-analysis" by means of *free associations à la* Freud, aiming at digging up one or more supposed suppressed "complexes," is a method of investigation which cannot be included in this book. It is a method that has to be confined to a certain number of specialists within the psychiatric and neurological world; the danger of suggestion (hetero-suggestion as well as auto-suggestion) is great in this method.

It has to be emphasized that the mental examination of the patient by no means is bound to elicit *any* anomaly that may exist. The patient may appear perfectly normal when interviewed—even at repeated interviews—yet his *behaviour* shows distinct anomalies. The possibility of a disorder of conduct must be kept in mind (Mercier). Continued observation during a longer period is necessary in these cases.

To make a complete examination of all *psychosensory and psychomotor functions* (collectively called psychosomatic functions) will be impossible as a routine examination to be employed in all cases. Only when there are indications of a disturbance of speech will a complete psychosensory-psychomotor examination be needed, and then so complete that it enables an exact *functional localization* diagnosis to be made. This examination is better performed apart from the rest of the neurological examination, for preference the day after admission, care being taken that the patient is not tired. The complete psychosomatic examination will be dealt with in the Appendix.

In the routine examination, then, no special tests for psychosomatic disturbances need be employed. If there is a disturbance of speech, one has first and foremost to ascertain whether it be due to an *articulatory* disturbance or not; the psychosomatic examination can then be carried out later (see Appendix, p. 159). As regards testing the articulation, see later, p. 50.

It has to be remembered, however, that aphasia and dysarthria may both be present together, and, in fact, motor aphasia is not unfrequently accompanied by some dysarthria.

2. CRANIAL NERVES.

I. The Olfactory Nerve.—On account of the frequent interference with the function of this nerve caused by insignificant local disturbances (rhinitis), the examina-

tion of its function has a limited value in the neurological clinic.

Either nostril is tested separately, the other one being closed by digital pressure. The use of ammonia, acetic acid, or similar substances should be avoided, as they also irritate the trigeminal nerve endings. Peppermint, camphor, and asafœtida may be used with advantage.

Anosmia may be caused by—

(1) Tumour in the frontal lobe.
(2) Cerebellar tumours.
(3) Atrophy of the olfactory nerves (tabes).
(4) Fracture of the floor of the anterior cranial fossa.
(5) Hysteria.
(6) Local lesions (*e.g.*, rhinitis).

II. **The Optic Nerve.**—In contradistinction to the olfactory nerve, the examination of this nerve is of extreme importance.

(α) *Acuity of vision* is tested by the ordinary test types (Schnellen).

(β) *Fields of vision*, either examined roughly, *à la* Donders, or, what is better, of course, by means of a perimeter.

Dr. Holth's portable perimeter is very handy and practical.*

In some cases the visual fields for different colours have a certain interest also. Normally, the visual field is larger for blue than for red, and larger for red

* For description of this perimeter see the *British Journal of Ophthalmology*, London, vol. iv., No. 10, October, 1920, p. 470.

than for green. In hysteria there may be a contraction of the visual field for one colour only—*e.g.*, for blue; one thus may find the visual field for red larger than for blue. A "spiral" visual field is a symptom of fatigue.

Homolateral hemianopsia occurs by lesion of the visual path behind the chiasma—viz., tractus opticus, capsula interna, occipital lobe. By lesion of the occipital lobe *quadrant hemianopsia* may be found; the visual cortex ("area striata") shows a distinct localization, the superior quadrants of the visual field corresponding to the infracalcarian, and the inferior quadrants to the supracalcarian region of the opposite side (Henschen, Gordon Holmes, P. Marie and Chatelin, Wildbrand).*

Bitemporal hemianopsia is due to a lesion of the chiasma—*e.g.*, pituitary tumour.

It is not until a pituitary tumour exceeds the precincts of the sella turcica and invades the middle cerebral fossa that the chiasma is involved.

Before this takes place signs of a disturbed pituitary function may be present: A *chromophile adenoma* (anatomically characterized by the predominance of eosinophile cells) may cause "pathological hyperpituitarism"—viz., acromegaly or gigantism (before ossification of epiphyseal cartilages), hyperglycæmia, glycosuria, increased metabolic rate, loss of sexual function; a *chromophobe adenoma* causes "pathological hypopituitarism"—viz., first and foremost depression of sexual function (amenorrhœa in women) and

* Probably the macula lutea chiefly corresponds to the posterior parts of the area striata (near the occipital pole), but probably also the macular fibres are distributed over a more extensive area of the visual cortex than are the fibres from other parts of the retina.

obesity ("dystrophia adiposo-genitalis"),* further diminished metabolic rate, and increased sugar tolerance.

Some pituitary symptoms (viz., polyuria, polydipsia, and disturbance of sugar tolerance) may possibly also be the result of a lesion of the infundibulum, the tuber cinereum, or adjacent brain areas.

(γ) *Ophthalmoscopic examination* is of extreme importance, and ought always to be included in the examination. On no account ought it to be omitted if the patient complains of headache. When the pupil is dilated by instillation of homatropine solution, it does not require much experience to ascertain the condition of the optic disc, which is the part of the fundus that chiefly interests the neurologist.

To get a maximal dilatation of the pupil it is advantageous to add 2 per cent. cocaine to the 1 per cent. homatropine solution. In individuals over thirty years of age it is to be recommended to follow up the examination with an instillation of eserin (salicylate of eserin in $\frac{1}{2}$ per cent. solution) in order to avoid any danger of glaucoma.

If such instillations are used, the pupils should be examined first, as a reliable examination of the pupils (their size and reactions, see p. 19) is impossible after instillation of homatropine, cocaine, or eserin.

The main points for the neurologist to decide are the presence of *optic atrophy*, of *papilloedema* or *choked disc*,

* A somewhat similar condition may in young patients be caused by hyperpinealism (obesity and retarded sexual development), whilst in children a destructive lesion of the pineal gland (hypopinealism) may cause *pubertas præcox* (or "macrogenitosomia præcox," hitherto only noted in boys).

and in order to decide these points attention must be paid to—

(1) The *colour* of the disc, normally pale pink; white or greyish-white in optic atrophy; red or greyish-red in papilloedema.

(2) The *border* of the disc, normally and in primary optic atrophy distinct and well defined; more or less blurred in papilloedema and secondary optic atrophy.

(3) Possible swelling of the disc. The swelling should be measured in diopters, indicated by the strongest + glass through which a clear image of the prominent disc can be obtained, as compared with strongest + glass (weakest −) through which the surrounding retina can be seen?

(4) The vessels. Are the arteries constricted? The veins distended? *Any hæmorrhages?* Any perivascular infiltration (greyish-white)?

(5) The lamina cribrosa, normally visible in the central cup, in primary optic atrophy visible right up to the edge of the disc; in papilloedema and secondary optic atrophy not visible at all.

It is sometimes difficult to decide whether the disc is paler than normal, and the initial stages of primary optic atrophy are therefore difficult to diagnose by means of ophthalmoscopy alone. Before any marked changes in the disc have occurred, however, a marked impairment of the vision is found (contraction of visual fields, diminished acuity of vision). The early diagnosis of primary optic atrophy will therefore as a rule chiefly be based upon the findings of the functional examination of the eye (visual fields, acuity).

Quite the contrary applies to papilloedema; here as a rule marked ophthalmoscopic changes precede any marked impairment of vision, which sometimes does not become manifest till secondary optic atrophy develops. *In the diagnosis of papilloedema, the ophthalmoscopic finding is therefore the deciding factor.* It consists of—

(*a*) Redness of the disc.

(*b*) Blurring of the edge of the disc, which can often not be clearly distinguished from the surrounding retina.

(*c*) Swelling of the disc, which ought if possible to be measured in diopters (indicated by the highest + glass that will give a clear image of the disc compared with the surrounding retina).

Normally, the temporal half of the disc is decidedly paler than the nasal half. The pathological pallor of the temporal halves, to which Strümpell attaches such importance for the diagnosis of disseminated sclerosis, is therefore difficult to decide and of little practical importance.

With regard to method, the direct is on the whole the easier; but when there is a high grade of myopia, or opacities, the indirect method is often the only one that can be employed. It would be best always to employ both methods: first the indirect for a general survey of the fundus, and then the direct for a more detailed examination of the disc.

III. **Nervus Oculomotorius.**
IV. **Nervus Trochlearis.**
VI. **Nervus Abducens.**

} Innervation of external and (III.) intrinsic eye muscles.

These nerves form in a way a functional unit, and therefore may be conveniently classed together in the examination. The examination of these nerves comprises the following points:

(A) *The Pupils.*—Their size. (In oculomotor lesions enlarged, mydriasis; in sympathetic lesions reduced, miosis.)* Give the diameter in millimetres. Are they equal? Are they regular in outline? Do they react to light directly? indirectly (consensual reaction)? Do they react to accommodation (convergence)? Do they react to pain (cilio-spinal reflex)?

When testing the *light reflex*, the simultaneous coming into play of the accommodation reflex must be avoided. In daylight the patient is asked to look through the window at some object in the distance, and to keep looking in this direction, whether his eyes be covered or not. Both eyes are shaded by the examiner's hands, and either eye alternately uncovered, pupillary changes being noted, both in the uncovered eye (*direct* reflex) and in the other (*consensual* reflex).

In artificial light the patient should look at the most distant corner of the room. A lamp or a candle is

* It has to be noted that the size of the pupils is influenced by the local application of *atropine* and *homatropine*, causing mydriasis due to paralysis of parasympathetic (oculomotor) nerve-endings in the sphincter pupillæ; *cocaine*, causing mydriasis, due to stimulation of sympathetic nerve elements innervating the dilator pupillæ; *eserin* (physostigmin) and *pilocarpine* causing miosis, due to stimulation of parasympathetic (oculomotor) nerve elements innervating the sphincter pupillæ. The size of the pupil is also altered in certain conditions of acute poisoning, being enlarged (mydriasis) in acute atropine, cocaine, and coniine poisoning—and reduced (miosis) in acute morphium, opium, and muscarin poisoning.

then held well out of the line of vision, so as to prevent the patient from fixing his eyes on it (accommodation reflex). The eye is then shaded and uncovered alternately, and the pupillary changes noted. For purposes of comparison it is always best to use the same intensity of light, which can only be obtained by means of artificial light held at the same distance from the eye. The reflex is said to be *good, sluggish,* or *absent,* according to the rapidity and extent of the contraction.

The *Argyll-Robertson sign* consists in absence of light reflex, the accommodation reflex being unaltered, a sign that is regarded as practically conclusive evidence of syphilis of the central nervous system—*e.g.*, tabes. It is one of the three classical signs of tabes: Argyll-Robertson's sign, Westphal's sign (absent knee-jerk), and Romberg's sign (see later).

During the last years the Argyll-Robertson sign has been found in several cases of *epidemic encephalitis* (" encephalitis lethargica ") without any evidence of syphilitic infection.

It has to be noted, however, that the pupillary light reflex may alter from time to time in one and the same person. Thus, *e.g.*, the reflex may be temporarily sluggish after a dose of alcohol, even after a comparatively small one, in some epileptics and people that are pathologically susceptible to alcohol.

The *accommodation reflex* is elicited by letting the patient alternately look at some distant object and fix some small object—*e.g.*, the tip of a pencil—held at the distance of some twenty centimetres from his eyes, and noting the corresponding change in the size of the pupil, which is kept slightly shaded.

In lesions of the *oculomotor nerve* the light reflex and the accommodation reflex are equally diminished or both absent. On the homolateral eye no light reflex (direct or consensual) can be elicited.

In lesions of the *optic nerve* it is only the light reflexes elicited from the homolateral eye (*i.e.*, homolateral direct light reflex and consensual light reflex *to* the other eye) which are diminished or abolished. From the contralateral eye one can still elicit an undiminished consensual light reflex in the eye, the optic nerve of which is affected, provided that the optic nerve of the other eye is not affected.

The *cilio-spinal reflex* is most easily elicited by pinching the skin of the patient's neck. When pain is elicited the pupil is seen to dilate. It is essential that the patient is really given a *painful* sensation; thus for the patient an unpleasant proceeding, which ought not to be repeated too frequently. It is important that the pupil during this test should be somewhat shaded, as in strong light the cilio-spinal reflex is often difficult to obtain (light reflex overpowering cilio-spinal reflex). The cilio-spinal reflex is regarded as a sympathetic reflex.

(B) *The Palpebræ.*—Is there any *ptosis* on one or both sides? Note how much of the cornea and eventually the pupil is covered by the eyelid. When in doubt, ask the patient to look up. In case of real ptosis he cannot raise his eyelids except by a compensating action of the frontalis muscle. If the eyebrows are fixed by the examiner's finger so as to prevent this compensatory action from occurring, the patient cannot raise the eyelids at all. When ptosis is pronounced, the frontalis has to be in a continuous state of contraction in order

to enable the patient to raise his eyes from the ground. The face in such cases assumes a peculiar expression, with continually wrinkled forehead and drooping eyelids. This is found in ophthalmoplegia (see Fig. 1) and also often in tabes, and constitutes in connection with miosis ("pin-point pupils") the chief characteristics of "facies tabetica."*

Ptosis is due to paralysis or paresis of the levator palpebræ, which is innervated from the oculomotor nerve. The levator has, however, an involuntary component, musculus tarsalis, innervated from the cervical sympathetic, which also innervates the musculus orbitalis Müller and the dilator of the pupil. Failure of this involuntary part of the levator also causes a slight ptosis, frequently called pseudo-ptosis.

The *cervical sympathetic syndrome* (Horner)—

1. Ptosis, due to paralysis of involuntary part of levator palpebræ.†

2. Enophthalmos, due to paralysis of musculus orbitalis (Müller).

3. Miosis, due to paralysis of musculus dilatator pupillæ.

For this latter cocaine is an irritant; cocaine instillation (2 per cent. solution) will therefore not cause any

* Sometimes I have noted that an obvious ptosis disappeared on voluntary exertion of the levator palpebræ—viz., when the patient was asked to "open his eyes wide," or to look upwards. In these cases the ptosis is probably to be ascribed to lack of tonic reflex innervation (hypotonia), and not to a true paresis of the levator palpebræ.

† According to Leriche and Fontaine, the ptosis seen after section of the cervical sympathetic is the result of an irritation ("un phénomène actif et non passif"). These authors insist upon the necessity of a revision of the physiology and pathology of the cervical sympathetic (*La Presse Médicale*, 1926, No. 84, p. 1313).

dilatation of the pupil when there is miosis due to a lesion of the cervical sympathetic.

4. Ipsolateral vasodilatation of head and neck (most easily seen in the conjunctival vessels), and

5. Ipsolateral anidrosis, are two less constant components of the syndrome.*

In contradistinction to this very distinct syndrome indicative of a lesion of the cervical sympathetic, the vagueness of the semeiology of the rest of the vegetative system is very noticeable.

At present two types of aberration from the average state of the vegetative nervous system may be distinguished:

1. Vagotonia (the "cold-blooded" type), with cold skin, bradycardia, and contracted pupils, with marked hypersensitiveness to hypodermic injections of pilocarpine (0·01 to 0·003 grm.).

2. Sympathicotonia (the "warm-blooded" type), with warm skin, rapid pulse, and dilated pupils, and with relative insusceptibility to pilocarpine and also to atropine (0·001 to 0·00065 grm.), while adrenaline (0·001 grm.) increases all the signs present.†

(C) *Ocular Movements.*—The patient is asked to look at the tip of the examiner's forefinger, or, better still, at

* As investigations of my ex-assistant, Dr. G. Raeder, have shown, the following three forms of the cervical sympathetic syndrome can be distinguished: (1) spinal—generally accompanied by spinal symptoms and signs (*e.g.*, atrophy of the intrinsic muscles of the ipsolateral hand). (2) cervical—the syndrome pure and simple. (3) the "paratrigeminal"—accompanied by trigeminal paresis and without vasomotor and secretory disturbances (*cf.* Figs. 6 and 7).

Cf. Norsk Magazin for Lægevidenskaben, 1918, No. 9, and *Brain*, 1924, vol. xlvii., Part II., p. 149, "Paratrigeminal Paralysis of Oculo-Pupillary Sympathetic."

† *Cf.* also the Appendix VI., "Pharmacological Tests of the Vegetative Nervous System," p. 194.

FIG. 1.—CASE OF COMPLETE OPHTHALMOPLEGIA WITH DOUBLE PTOSIS AND COMPENSATORY CONTRACTION OF THE FRONTALIS MUSCLE.

FIG. 2.—CASE OF INCOMPLETE RIGHT OCULOMOTOR PARALYSIS.

The patient is trying to look up towards the ceiling. The paralysis of the right superior rectus is clearly seen.

FIG. 3.—THE SAME PATIENT AS IN FIG. 2.

The patient looks down. Although there is no facial paralysis, the upper eyelid does not follow the movement of the eye on the affected side.

FIG. 4.—CASE OF CEREBELLOPONTINE TUMOUR WITH A RIGHT FIFTH CRANIAL NERVE LESION.

The patient opens her mouth and the lower jaw is seen to deviate towards the affected side.

To face page 24.

the point of a thin pencil. This is then moved to the right and left, upwards and downwards, whilst the patient tries to follow it with his eyes.

The examiner notes whether a squint occurs (see Fig. 2), and the patient is made to indicate if in any position he sees two images of the pencil.* In this case it is noted in which position the diplopia occurs, the relative position of the images to one another, and, finally, one eye is covered suddenly and the patient asked which image has disappeared (the top one or the lower one, the right or the left one). This the patient often will have some difficulty in deciding. If a red glass be put in front of one eye it facilitates matters considerably; the patient will then easily be able to describe the relative positions of "the red pencil" and the white pencil. (For this method a white or yellow pencil, a match, or a lighted candle is the best test object.)

It is also noted whether the patient lifts his upper eyelids when looking up, or must use the frontalis muscle to raise his eyelids (latent ptosis, *cf.* above); and if, again, he lowers his eyelids when he looks down (*failure* to do this, Gräfe's sign, is fairly frequent in Graves' disease).†

Movement of the patient's head during these tests must be prevented; to insure this it is best for the examiner to put his left hand on the patient's head.

A normal individual, when asked to look to the right or left, will naturally turn his head in that direction. The

* In ocular paralysis of long standing the patient sometimes learns to suppress the image derived from one eye. Then, of course, diplopia does not occur.

† Sometimes this is also seen in incomplete oculomotor paralysis (see Fig. 3). The explanation of this phenomenon is debated.

eyes are also slightly turned, but the normal individual chiefly relies on the movement of the head for bringing the desired object into focus.

In *paralysis agitans* this is very often different; here, when the patient is asked to look to the right or left, the eyes are moved *ad maximum* and the head but very little, or at least very slowly; the resemblance to a person who is wearing a too tall and too narrow collar is striking. This sign of *rotary neck rigidity* is a characteristic sign of paralysis agitans. It need hardly be added that it is not pathognomonic, as it also occurs in other conditions—*e.g.*, cervical spondylitis, torticollis rheumatica.

Nystagmus is a very important sign, and has to be carefully looked for. It is an involuntary movement of the eyes which chiefly occurs in lateral deviation, and consequently it will be observed when the patient is looking to the right or left. In the above tests for ocular movements it is important to let the patient *keep looking* to the right and to the left for five or six seconds; nystagmus will then, if present, as a rule be readily discovered.

Extreme deviation should, however, be avoided, as this normally not seldom elicits nystagmus or nystagmoid jerks.

Various forms of nystagmus are distinguished—viz.:

(1) *Horizontal* (the commonest form);
(2) *Vertical* (the rarest form); and
(3) *Rotatory*

—all according to the direction of the movement. We also distinguish between a *rhythmical* and an *oscillating* form. The *rhythmical* form has a slow

movement in one direction succeeded by a quick movement in the other direction. When looking to the right, a horizontal rhythmical nystagmus thus consists of one slow movement to the left succeeded by a quick movement to the right. This is the sort of nystagmus which can be seen in passengers in a train or a bus when they are looking out on the stationary surroundings (a very instructive observation for the beginner);* in the *oscillating* form the two movements are equally fast.

The nystagmus is usually labelled according to the direction in which the patient has to look for the nystagmus to occur (this in rhythmical nystagmus, as a rule, corresponds to the direction of the quick motor component). "Nystagmus to the right," then, means: nystagmus occurring when the patient is looking to the right.

Slight rotary nystagmus is often difficult to discover. When looking for it, it is best to keep the eye on a bloodvessel in the conjunctiva bulbi and note if this shows any movement.

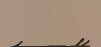
Care is necessary in the use of the term "nystagmus"; some observers are apt to find nystagmus in nearly every patient they examine. Nystagmoid jerks may occur in a tired patient when trying to fix an object to the right or left which requires extreme deviation of the eyes (*cf.* above). A slight unsteadiness of the

* A similar nystagmus is also developed when a revolving drum with vertical lines is looked at. This may be utilized in deciding if a little baby, a speechless patient, or a simulant is blind or not. If nystagmus occurs on rotating such a drum in front of the patient, it follows that the patient can see.

eyes must not be put down as nystagmus; it may be entirely due to lack of attention on the part of the patient. When in doubt, "nystagmoid jerks" is the most neutral term for recording the finding.

For recording the findings it is convenient to indicate the direction of nystagmus by means of arrows in the following way:

horizontal nystagmus to the left (the direction of the arrow corresponding to the position of the patient facing the observer).


horizontal nystagmus to the right.

vertical nystagmus upwards.

„ „ downwards.

rotary „ to the left.

„ „ to the right.

horizontal and rotary nystagmus to the left, etc.

The patient's power of convergence (often diminished in Graves' disease) must also be tested, if this has not already been done when examining the pupillary reflex on accommodation.

Oculogyral spasms must also be noted. These are attacks of conjugate ocular deviation in one direction—most commonly upwards. They may by some patients at least be voluntarily surmounted for a moment (the effort usually being accompanied by strong palpebral tremor). The attacks may last from a few minutes up to half an hour or more. They occur in post-encephalitic conditions

The diagram, Fig. 5, gives an approximate idea of the isolated action of the different muscles.* The rectus superior, the rectus inferior, and the obliqui also rotate the eye round an antero-posteral axis, so that in paralysis of these muscles the double images are not parallel.

For a more detailed description of the function of the eye muscles the reader must be referred to the textbooks on ophthalmology.†

The most common of all ocular paralyses is the abducens paralysis affecting the rectus externus. This may occur isolated as a symptom of increased intracranial pressure, and also as the result of pressure

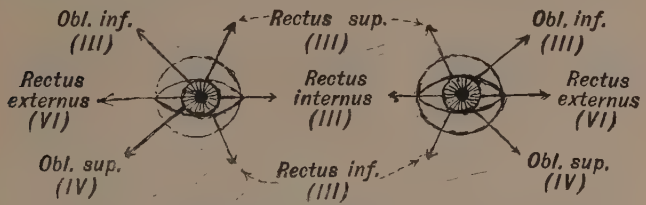


FIG. 5.—DIAGRAM OF THE ACTION OF THE DIFFERENT EXTERNAL EYE MUSCLES.

from distended arteriosclerotic bloodvessels on the basis cerebri; therefore it has only a limited value for the focal diagnosis.

Interesting is the distinction between the *nuclear* and *infranuclear* paralysis of the abducens. The abducens nucleus is, if not identical with, at any rate in very close

* *In vivo*, however, no individual eye muscle has an isolated action. The ocular movements are the result of co-ordinated action of *all* eye muscles (*cf.* Appendix, "On Diplopia," p. 170).

† An admirable description of the function of the eye muscles is to be found in Professor Schiøtz' little book on that topic in Norwegian.

relation to, the centre for the conjugate movement of both eyes to the corresponding side.

In an affection of the sixth nucleus, therefore, the patient will be unable to look to the side of the lesion.* That in such cases the rectus internus on the other side is not paralyzed may be readily demonstrated by trying the patient's convergence, whereby both recti interni are seen to act well.

Supranuclear lesions always affect both eyes. It may here be mentioned that all the motor cranial nerves, except the twelfth and the lower part of the seventh, have a bilateral cortical innervation.

Isolated paralysis of rectus internus points to a partial lesion of the oculomotor nucleus (often seen in epidemic encephalitis).

V. **The Trigeminal Nerve**—(*a*) The *Motor Division*, innervating all muscles of mastication, masseteres, temporales, pterygoidei, is tested in the following way:

The patient is asked to clench his teeth as hard as he can, and at the same time the masseter and temporalis muscles are tested by palpation.

The patient is further asked to open his mouth wide, and any deviation of the lower jaw is noted. In affections of the motor part of the trigeminal the lower jaw deviates to the side of the lesion, on account of the deficient action of the pterygoidei muscles† (*cf.* Figs. 4, 7, and 8).

In order to be exact, the teeth should be used as

* In lesions of the region of the quadrigeminal bodies (*e.g.*, in pineal tumours) a similar phenomenon is met with—viz., inability or difficulty of the upward conjugate movement of the eyes.

† These normally pull the mandibular condyles forward during the opening of the mouth. By palpation just in front of the tragus the condyles can easily be felt moving forwards.

FIG. 6.—LEFT "PARATRIGEMINAL" SYMPATHETIC LESION. PTOSIS AND MIOSIS ON LEFT SIDE.

(Raeder: *Brain*, vol. xlvii., part ii.)

FIG. 7.—LEFT TRIGEMINAL PARALYSIS.

Lower jaw is seen to deviate to the left when patient (same as in Fig. 6) opens his mouth.

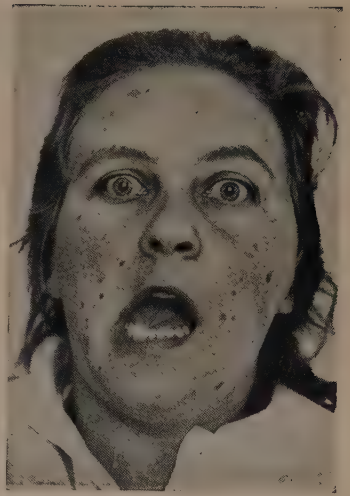


FIG. 8.—RIGHT TRIGEMINAL PARALYSIS.

When the patient opens her mouth the jaw deviates to the right.

To face page 30.

guide when judging the deviation. The interval between the two median incisors corresponds approximately to the middle line, but does not always correspond exactly in the two jaws. Note the relative position of the teeth in the upper and the lower jaw when the mouth is closed, and see if any deviation from this occurs when the mouth is opened. Where there is a facial palsy it often looks on opening the mouth as if there is a deviation of the lower jaw, although no such deviation takes place. Looking at the relative position of the teeth in such cases readily undeceives the observer. When in doubt palpate the movement of the condylar* process of the mandibula, when the mouth is opened, and also ask the patient to move his lower jaw side-ways. In trigeminal lesion there is diminished or no excursion to the opposite side, on account of the deficient action of the pterygoid muscle on the paralyzed side.

(*b*) *Sensory Division.*†—Sensation of the face and a varying portion of the scalp has to be tested—

(1) By means of a piece of paper or cotton-wool—touch.
(2) By means of pin-pricks—pain.
(3) By means of test-tubes filled with hot and cold water—temperature.

As regards the technique the reader is referred to the section dealing with examination of superficial sensation (see p. 79).

* The normal forward movement of the condylar process is missing on the paralyzed side.

† A number of interesting anatomical, clinical, and experimental data will be found in M. W. Gerard's article, "Afferent Impulses of the Trigeminal Nerve," *Archives of Neurology and Psychiatry*, March, 1923, vol. ix., No. 3, p. 306.

The fibres conducting *pain* and *temperature* sensation descend in the spinal tract and pass at various levels to the cells of the *spinal nucleus*; the fibres conducting *tactile* impulses pass to the cells of the main sensory nucleus in the pons.

The impulses of deep sensation are not carried by the trigeminal, but by the facial nerve.

The sensory part of the trigeminal nerve is connected with the contralateral hemisphere only; the motor part with both.

The points of exit of the chief sensory nerves are also tested for tenderness (frontal, supraorbital, infraorbital, and mental nerves). In trigeminal neuralgia these are often excessively tender even between the neuralgic attacks.

The *corneal reflex* (constant in all normal individuals) and the conjunctival one (inconstant) may conveniently be examined here (a joint function of the fifth and seventh cranial nerves; for description, see pp. 106-107).

Loss of corneal reflex is often an early sign of the trigeminal (and facial) affection encountered in cerebello-pontine tumours. Sometimes it remains the only sign of trigeminal (and facial) involvement.

VII. **The Facial Nerve.**—Has the patient's face a one-sided appearance? If so, this may be due to paresis or paralysis of the one side, to contracture of the other, to a developmental asymmetry, or to unilateral facial atrophy.

In order to obtain further information the following tests have to be applied:

(1) The patient is asked to raise his eyebrows;

(2) To frown; and

FIG. 9.—RIGHT FACIAL PARALYSIS OF THE ORDINARY PERIPHERAL TYPE.

Upper and lower portion of facial musculature equally affected. Patient is trying to shut her eyes and show her teeth (lagophthalmos and Bell's phenomenon).

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FIG. 10.—RIGHT FACIAL PARALYSIS OF ORDINARY PERIPHERAL TYPE.

Upper and lower portion of facial muscles equally affected. Patient is trying to close her eyes and pull back the corners of her mouth (lagophthalmos and Bell's phenomenon).

To face page 33.

(3) To shut his eyes. He is asked to screw them up as tightly as he can; by then trying to open them against the patient's effort to keep them closed, an impression of the power of the orbiculares oculorum is obtained.

When there is *lagophthalmos* (*i.e.*, the eye remains open in spite of the patient's effort to shut it), the eyeball will be seen to move upwards (and slightly outwards), whereby the cornea is being covered by the upper lid (*Bell's phenomenon*).

This phenomenon is an involuntary synkinetic movement which is normally present, and which persists (perhaps even becomes exaggerated) in supranuclear paralysis of both recti superiores, as observations by my first assistant, Dr. Saethre, tend to show.

(4) He should also be asked to shut each eye separately. (Inability to shut one eye alone is in many cases the only remainder of an old facial palsy; it may also be the only defect in the upper facial musculature in central [pyramidal] facial paresis.) It should also be noted if synkinetic movements during this occur, and if these are symmetrical in their occurrence.

The above are tests for the muscles of the upper facial portion, which has a bilateral cortical innervation, and which consequently is not affected in supranuclear lesions—*e.g.*, the ordinary capsular hemiplegia—or at least not to the same extent as the lower part of the facial muscles. These are tested in the following manner:

(5) The patient is asked to show his teeth;
(6) To blow out his cheeks;
(7) To whistle; and
(8) To smile.

Many patients—most females—smile spontaneously after the effort of whistling. It is important to take note also of this and the patient's spontaneous emotional expressions in general.

In thalamic and lenticular lesions it is often found that there is deficiency or loss of the movements of emotional expression in the opposite side of the face, whilst the above tests (all carried out on the examiner's command, and having nothing to do with emotional expression) betray no disturbance (*cf.* Figs. 19 and 20).

On the other hand, it is commonly found in organic lesions (*e.g.*, hemiplegia), which have affected the pyramidal fibres to the facial nucleus, that whilst the patient's voluntary movements (as evidenced by his response to tests 5 to 7) are decidedly paretic, yet the emotional innervation (as evidenced by the *spontaneous* smile) does not betray any paresis, and may even, as the author has shown,* be accelerated and exaggerated (*cf.* Figs. 11, 12, 13, and 14, 15, 16).

This facial dissociation (voluntary movements paretic whilst emotional movements less paretic, intact or exaggerated) is indicative of a *central* lesion.

In *peripheral* facial palsy any such marked dissociation is never seen. Here voluntary (or intentional) and emo-

* *Cf.* Monrad-Krohn: "On the Dissociation of Voluntary and Emotional Innervation in Facial Paresis of Central Origin," *Brain*, 1924, vol. xlvii., Part I.

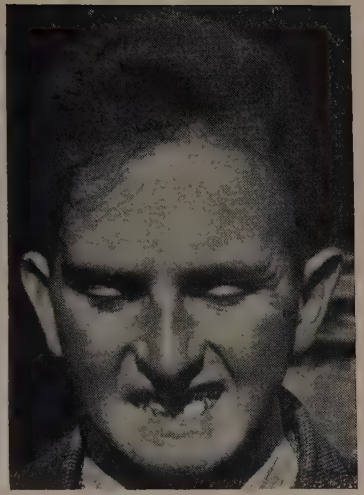

FIG. 11.

FIG. 12

FIG. 13.

CASE OF HEMIPARESIS DEXTRA OF TRAUMATIC ORIGIN: ACCELERATION AND EXAGGERATION OF EMOTIONAL MOVEMENTS ON RIGHT (PARETIC) SIDE OF FACE.

(Figs. 11, 12 and 13 are reproductions from kinematographic film. Monrad-Krohn: "On the Dissociation of Voluntary and Emotional Innervation," *Brain*, 1924, vol. xlvii., part i., p. 22.)

FIG. 11. — The patient shows his teeth Marked right facial paresis is seen.

FIG. 12.—The patient *begins* to smile at a joke. The smile starts on the right (paretic) side.

FIG. 13.- The patient is smiling at a joke. Facial movements more pronounced on the right (paretic) side.

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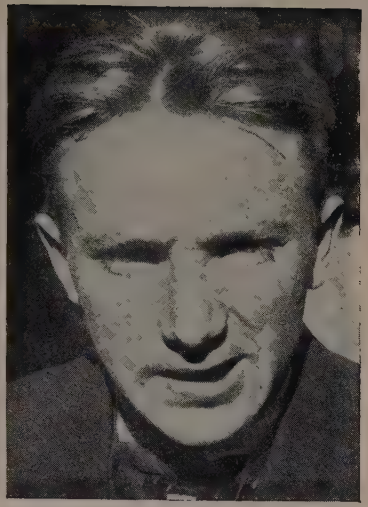

FIG. 14.

FIG. 15.

FIG. 16.

TRAUMATIC LESION OF THE LEFT HEMISPHERE INVOLVING THE FACIAL MOTOR AREA (CORTEX OR SUBCORTICAL REGION) PRODUCING A PARESIS OF VOLUNTARY MOVEMENTS, BUT AN ACCELERATION AND EXAGGERATION OF THE EMOTIONAL MOVEMENTS OF THE RIGHT SIDE OF THE FACE.

(Reproductions from kinematographic film. Monrad-Krohn: "On the Dissociation of Voluntary and Emotional Innervation," *Brain*, 1924, vol. xlvii., part i., p. 22.)

FIG. 14.—The patient tries to show his teeth. Distinct voluntary paresis on right side.

FIG. 15.—The beginning of a spontaneous smile. Emotional acceleration on right side.

FIG. 16.—A fully developed spontaneous smile. Exaggeration of emotional innervation on right side.

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FIG. 17.

FIG. 18.

HEMIPARESIS SINISTRA: TUMOUR IN POSTERIOR PART OF RIGHT FRONTAL LOBE.

FIG. 17. — The patient shows his teeth. Marked left facial paresis is seen.

FIG. 18. — The patient is exerting all his strength in trying to pull his right hand away from the observer. During this the involuntary synkinetic facial movement is much stronger in the paretic left side than on the right.

FIG. 19.

FIG. 20.

EMOTIONAL PARESIS OF LEFT SIDE OF THE FACE (POST-ENCEPHALITIC PARKINSONIAN STATE).

(Monrad-Krohn: "On the Dissociation of Voluntary and Emotional Innervation," *Brain*, 1924, vol. xlvii., part i.)

FIG. 19.—Patient shows his teeth; no pronounced asymmetry.

FIG. 20.—Spontaneous smile; marked emotional paresis on left side.

To face page 35.

tional movements always suffer to the same extent. Only in a few cases have I seen emotional movements that seemed to be slightly stronger (*i.e.*, slightly less paretic) than the voluntary ones; but I have never seen the reverse, and never have I seen more than the slightest suggestion of this difference between voluntary and emotional movements in peripheral facial palsy.

Some patients (particularly lady patients) with slight facial palsy complain that their facial palsy only (or chiefly) becomes apparent when they smile a true emotional smile ("when they cannot help smiling"). These patients have learned to subdue their voluntary movements and keep them within the range where the asymmetry of their face does not show, whilst their emotional smile cannot be subdued to the same extent. When testing the *maximum* extent of their voluntary movements (*e.g.*, asking them to pull the corners of their mouth as far back as possible), one finds the same amount of paresis for voluntary movements as for the smile; in other words, in these cases there is only an apparent but *no real* dissociation between emotional and facial movements.

The above-mentioned dissociation is often rather bewildering to the beginner. There is also another phenomenon sometimes seen in supranuclear facial paresis which may be rather confusing. This is illustrated in Figs. 17 and 18. It happens that the automatic synkinetic movement, which normally occurs in the facial region on great bodily exertion, becomes exaggerated in the paretic side (Fig. 18).

The stiff, masque-like expression in *paralysis agitans* is characteristic; if a book or a piece of paper be held in front of the patient's mouth, thus excluding it from view, it will not be possible to judge by the rest of the patient's face whether he is speaking or not. It has to be kept in mind, however, that a bilateral facial paresis (as seen, *e.g.*, in encephalitis lethargica) may give a similar masque-like facies, which in pathogenetic respect

is quite different from the real paralysis agitans facies of lenticular origin (also seen in parkinsonoid forms of encephalitis lethargica).

Uncontrolled attacks of crying and laughing occur in many nervous diseases, notably in pseudo-bulbar paralysis. Characteristic also is the absolutely blank expression which we encounter in dementia præcox, interrupted now and again by smiles for no apparent reason—probably in response to hallucinations.

In the above tests it is important to note, not only any deficiency of movement, but also if the movements are accompanied by any tremor. *Perioral fibrillary tremor* is a sign that is frequently and typically found in *dementia paralytica*, often very early in the development of the disease.

Tremor palpebrarum is a frequent finding also in purely "functional" conditions, and has not the same significance as the perioral tremor. Any over-action of the facial movements ought also to be noted.

Also other involuntary movements may be seen in the facial region—*e.g.*, various forms of tic (motor obsessions, *cf.* p. 8), myoclonic twitchings (in myoclonic forms of epidemic encephalitis), post paralytic facial spasms. It is of interest in these post paralytic spasms to note the time relation of the spasm to the normally occurring blinking of the eyes.

If the spasm is synchronous with the ordinary blinking, which normally occurs simultaneously in both eyes from time to time, it is regarded by some clinicians as due to fibres intended for the orbicularis oculi having gone astray, during the process of regeneration, to other facial muscles.

Lesions of the Facial Nerve.—1. Lesion of the facial trunk peripheral to the chorda tympani's leaving the nerve: paralysis of the *whole* corresponding side of the face, the upper facial portion included.

In *leprosy* we meet with another and peculiar type of peripheral facial paralysis due to lesions of the most peripheral (terminal) *branches* of the facial nerve, generally on both sides. The chief characteristics of these facial paralyses are—

(*a*) The upper part of the face—particularly the orbicularis oculi and the corrugator—is more often and more intensely affected than the lower part of the face.

(*b*) This paralysis of the upper part of the face is, as a rule, bilateral.

(*c*) In the inferior part of the face the supraoral muscles are more commonly affected than the infraoral muscles.

(*d*) This paralysis in the inferior part of the face is not so constantly bilateral as the paralysis in the upper part of the face.

(*e*) The paralysis is accompanied by extreme hypotonia or atonia, which causes ectropion of the eyes in a great number of cases—and in some cases where the orbicularis oris is equally affected, " ectropion of the mouth." This is generally accompanied by dribbling of saliva from the mouth.

It is a striking feature of the facial paralysis in leprosy that the individual muscle is often affected in a variable degree in its different parts. Thus it is not rare to see the frontalis muscle strongly paretic or even paralytic in its medial part, whilst the more lateral parts of the muscle have retained a comparatively good function. When in such a case the patient is made to raise his eyebrows, this often results in a characteristic reaction consisting of an elevation of the lateral parts of the eyebrows only.

When the orbicularis oris is affected, a peculiar configuration of the mouth, highly characteristic of leprosy, often occurs. Corresponding to some of the radial muscles of the mouth (*e.g.*, zygomaticus), which have not yet suc-

cumbed to the paralysis and which have retained their normal tonus, a retraction is seen in the lips, which are otherwise flabby, swollen, and " ectropic "* (*cf.* Fig. 21).

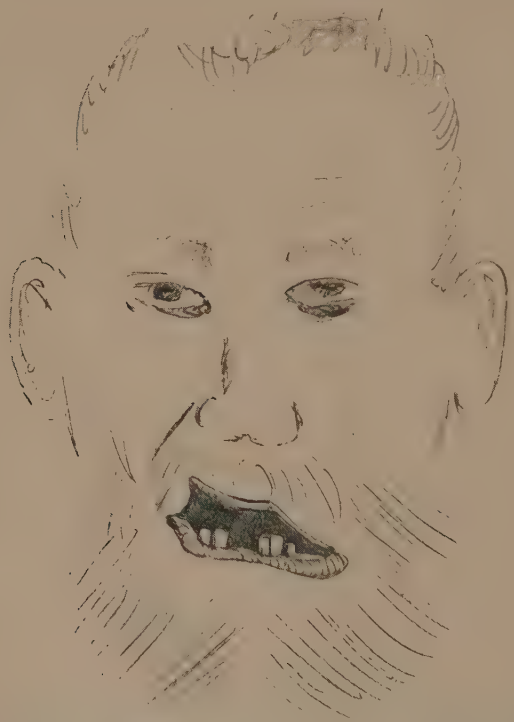

FIG. 21.—ROUGH SKETCH OF OLD LEPROSY CASE WITH TYPICAL FACIAL PARALYSIS.

The mouth shows a typical defiguration. Only on the right side is the naso-labial fold still present. On this side also a retraction of the upper lip is seen. In the frontal muscle only the most lateral parts have retained their mobility. Ectropion on both sides. (Monrad-Krohn: "The Neurological Aspect of Leprosy," Oslo, 1923.)

* *Cf.* Monrad-Krohn: " The Neurological Aspect of Leprosy," Oslo, 1923.

These irregular facial paralyses are so typical of leprosy, and of such frequent occurrence* in this disease, that the practitioner in leprosy districts ought to be thoroughly familiar with them, and always on the look-out for them in suspect cases.

2. Lesion central to the departure of chorda tympani, but peripheral to ganglion geniculi:† paralysis of the whole corresponding side of the face, as in (1), and besides *loss of taste in the anterior two-thirds of the corresponding side of the tongue.*

3. Lesion between ganglion geniculi† and the pons: paralysis of the whole corresponding side of the face, as in (1); no disturbance of taste, as in (2), but as a rule some deafness, as the acoustic nerve is generally affected. If the acoustic nerve is intact hyperacusis as a rule obtains, on account of stapedius paralysis (thus in any case a disturbance of hearing).

4. Lesion in the pons itself, nuclear or infranuclear: paralysis of the whole corresponding half of the face, but without any disturbance of taste or hearing. As the fibres of the facial nerve, however, come into close relationship to the nucleus and the fibres of the sixth nerve, a facial paralysis of pontine origin is, as a rule, accompanied by abducens paralysis (nuclear or infranuclear; *cf.* under Third, Fourth, and Sixth Cranial Nerves, pp. 29 and 171).

5. Supranuclear lesion: paralysis never complete, and chiefly or exclusively confined to lower portion of

* In a series of fifty-nine cases of leprosy I have found facial motor paralysis in forty-two cases—*i.e.*, in 71·2 per cent. (*loco citato*).

† Inflammation of the *geniculate ganglion* itself produces pain and *herpes of the auricle* and external auditory meatus.

the face, the upper portion being either intact or much less affected. As the lesion is practically always situated above the decussation of the pyramidal fibres to the seventh nucleus, the paralysis is found on the opposite side of the lesion.

(Reaction of degeneration occurs only in the first four forms, not in (5); *cf.* later under Electrical Examination, pp. 126, 133, 137.)

The examination of *taste* ought always to be carried out in a case of facial paralysis. The patient is asked to put out his tongue and to keep it out during the test. A little powdered sugar, salt, citric acid, and quinine is then placed successively on the tongue, and rubbed in with a corner of a towel or a piece of cotton-wool. The patient must then indicate the sensation he gets by pointing to one of the following words which have been written up for him:

Sweet.
Salt.
Sour.
Bitter.

He must keep the tongue out till he has indicated the sensation, as otherwise the substance may be carried on to other parts of the tongue than those it is wished to test (posterior third or the other side). Each half of the tongue has to be tested separately, and the results compared. The reaction can be timed. The taste varies a great deal in normal persons, particularly in persons who smoke heavily.

The examination does not form an integral part of the ordinary routine examination, but when under-

taken it ought to be carried out exactly as indicated above. The use of solutions instead of powdered substances is not to be recommended, since it is then more difficult to keep the stimulus localized to the part being tested.

It is only the anterior two-thirds of the tongue (*i.e.*, the part innervated by the chorda tympani fibres) that can be tested in this way. For the posterior third (*cf.* glosso-pharyngeus) only the galvanic test can be applied. This can, of course, also be applied to the anterior two-thirds, and will therefore briefly be described here. A wire electrode of copper is used and a weak galvanic current (0·2 to 0·4 milliampère). The wire electrode is used as anode and applied to the tongue, eliciting a peculiar, acid taste. By this method the extension of any disturbance of taste can be fairly distinctly mapped out.

The facial nerve also carries the impulses of *deep sensation* in the face (at least, the impulses of deep pressure pain, the chief quality of deep sensation to be examined in the facial region).* For the examination of the deep pressure pain in this region an algometer is very useful, if not absolutely necessary.

Probably the facial nerve also contains sympathetic fibres. The easier regeneration of these and a supposed subsequent state of irritation of these same fibres (which are believed to innervate the sarcoplasm of the facial muscles), should, according to Louisa Levi,† be the cause of the facial contracture so often seen after facial palsy.

* *Cf.* Loyal E. Davis: "The Deep Sensibility of the Face," *Archives of Neurology and Psychiatry*, March, 1923, vol. ix., No. 3, p. 283.

† *Cf. La Presse Médicale*, 1924, ii., No. 67, p. 667.

VIII. **The Acoustic Nerve.**

(α) *The Cochlear Nerve.*—It is noted at what distance from either ear the patient can hear an ordinary watch. Any difference between the two sides is to the neurologist of more importance than the absolute value of acuity of hearing. If any difference is found, it is important to compare air and bone conduction by placing the tuning-fork with the branches close to the ear (air-conduction) and with the handle on the mastoid process (bone-conduction), asking the patient which he hears loudest. The time by which the one sensation outlasts the other can also be measured, *à la Rinne.* The tuning-fork is then first placed on the mastoid process, and when the patient cannot hear any more in this position it is held with the branches close to the concha, and the time measured till the patient indicates that he hears nothing more.

Normally the hearing by air-conduction is stronger and lasts longer than hearing by bone-conduction.

In deafness due to lesions of the labyrinth and the nervous apparatus this normal relation is maintained; but in middle-ear lesions and blocking of the meatus externus the relation between air and bone conduction is reversed, the hearing by bone-conduction being stronger and outlasting the hearing by air-conduction.

In this case it is, of course, necessary to perform otoscopy, which, however, ought not to be neglected in any case of disturbance of hearing.

(β) *The Vestibular Nerve.*—The vestibular apparatus is in functional respect closely bound up with the cerebellum. The vestibulo-cerebellar apparatus is of

great importance for the maintenance of equilibrium as well as for co-ordination in general. This will be further dealt with under the heading Co-ordination; here the ordinary vestibular tests only will be mentioned.

As the tests take a fairly long time and are apt to tire the patient, thus rendering him unfit for the rest of the examination, these vestibular tests had better not be made as part of the first examination, but can more conveniently be carried out as a supplementary examination one or two days later. The vestibular examination comprises the following tests:

1. Rotation test.
2. Caloric test.
3. Galvanic test.

In the two former the stimulus is provided by currents set up in the endolymph of the semicircular canals.

In the third the stimulus consists of an electrical irritation of the nervous elements of the vestibulo-cerebellar apparatus.

As a response to vestibular irritation we normally find—

(*a*) Nystagmus.

(*b*) Involuntary deviation of the patient's position —postural deviation (tendency to fall to one side in Romberg position).

(*c*) Involuntary deviation of the movements— kinetic deviation (" past-pointing ").

For eliciting the two latter it is necessary to let the patient shut his eyes.

These various forms of reaction generally correspond to each other.

1. For the *Rotation Test* the patient is placed on a rotating chair with a head-rest, which enables the head to be fixed in the desired position, so as to bring one particular pair of semicircular canals into play.* In order to obtain pure horizontal nystagmus the head is tilted slightly forward.

The patient is rotated ten times quickly in succession (duration about twenty seconds), and then suddenly stopped. During the rotation nystagmus in the direction of the movement occurs, but for obvious reasons this escapes observation.

When the rotation stops, nystagmus in the opposite direction occurs, and this is easily observed by making the patient look about 45 degrees to the opposite side of that in which rotation has taken place. This nystagmus is accompanied also by other phenomena. If the patient be made to walk in a certain direction with his eyes shut, a noticeable deviation occurs to the opposite side of the nystagmus, and by testing directional movements of the limbs with the eyes shut all movements of the limbs show the same deviation. The most practical way of testing for deviation is to let the patient point with the whole arm rigidly extended, all movements taking place at the shoulder-joint; on account of its construction, movements at this joint show deviation more readily than movements at the elbow-joint. The patient is asked to point to the observer's finger, which is held in front of the patient

* The use of an ordinary piano stool must be avoided. If there is not sufficient rest for the back and head, the feeling of giddiness may reach a very unpleasant degree and even result in collapse.

at such a distance that with his arm extended horizontally he just reaches it with his forefinger. He is then asked to shut his eyes, let his arm sink right down, and then again raise it so as to make his forefinger meet the observer's (*cf.* pointing tests under Special Examination for Cerebellar Signs, p. 76).

When there is a double vestibular lesion, the reaction described above is deficient or wholly absent. To examine either vestibular apparatus separately is not possible by this test. For this purpose other tests must be used.

2. *The Caloric Test.*—The external meatus is syringed with hot or cold water (50° to 55° C. and 18° to 25° C.), whereby currents are set up in the endolymph on the thermo-siphon principle. The great advantage of the test is that it is *a unilateral* test—viz., either vestibular apparatus is tested separately, and even one particular semicircular canal can be singled out for examination by placing the head in different positions.

The most practical plan is to examine the patient in the horizontal position, face upwards; in this position a horizontal nystagmus is elicited, which is much easier to observe than the rotary.

It has to be borne in mind that the caloric reaction is diminished, even abolished, when the meatus is clogged with cerumen,* and also when the middle ear is filled with blood-clot; on the other hand, retraction of the drum accentuates the reaction. It may in this connection be mentioned that vestibular vertigo may be

* The caloric test ought, therefore, always to be preceded by otoscopy. If the drum is perforated, the caloric test ought to be omitted.

caused by unilateral alteration of the air-pressure in the middle ear (Scott).

Brünings has constructed an apparatus for the caloric test which may well be used; but it has to be borne in mind that *it is not an exact quantitative test,* even with this apparatus. The whole question to decide is: "Is any response obtained or not?" And for this purpose an ordinary ear syringe of 100 c.c. capacity is sufficient. Three or four syringes in quick succession will generally suffice to elicit reaction.

During the last year I have used *cold air* instead of cold water for the caloric tests and find this considerably more convenient and just as reliable. It has the advantage, besides, that it can also be used where there is perforation of the drum.

The apparatus used is devised by Dundas Grant and is quite simple. The air is blown through a spiral-shaped metal pipe of about 2 millimetres diameter and about 30 cm. long, covered with cloth or some other porous tissue on to which ethyl chloride is sprayed or poured. The evaporation of this is sufficient to cool the air blown through the pipe by means of an ordinary rubber balloon.

The effect of this cold air irritation is exactly the same as that elicited by cold water irrigation.

3. The *Galvanic Test* can also be employed unilaterally by placing a round flat electrode of 6 to 7 centimetres diameter as *anode* on the ear and the kathode in the patient's hand on the same side.

The patient stands in the Romberg position—*i.e.*, feet close together, eyes shut.

By suddenly closing a current of 5 to 7 milliampères the patient is drawn over to the side of application (also slightly rotated to the other side and slightly drawn backwards). Deviation in walking and pointing may also be elicited by this test.

The postural deviation, as described, may occur by a current of 2 milliampères—commonly 6 to 8 milliampères are required; but in some patients a current of 15 to 20 milliampères has to be employed in order to elicit reaction. If this does not elicit any response, the test is reckoned to be negative.

The galvanic test may be positive, even though the whole labyrinth be destroyed, as long as the vestibular nerve ganglion and the more central parts of the vestibulo-cerebellar apparatus are intact.

IX. The **Glosso-pharyngeal,** and

X. The **Pneumogastric** nerves are generally recorded together, as their areas of innervation seem to overlap —at least, they cannot be clearly distinguished.

The patient is made to open his mouth and say "Ah." The raphé palati is hereunder watched. In cases of one-sided paresis it deviates to the non-paralyzed side.*

The patient is made to swallow, and the power with

* Research has shown that the innervation of the soft palate is rather complex. The *tensor veli palati* is supplied by the fifth cranial nerve. A paralysis of this muscle is difficult, if not impossible, to recognize clinically. The *levator veli palati* is supplied by the bulbar portion of the eleventh cranial nerve, which is in reality only the caudal portion of the tenth nerve. As this is the predominant palatal muscle we still continue to place this test under the heading of the tenth cranial nerve.

which the larynx is pulled upwards during the act of deglutition is tested. A convenient method is to let the patient sip from a glass of water. Notice if any regurgitation through the nose takes place. Is it accompanied by coughing ?

Laryngoscopic examination ought always to be performed if there are any other signs of affection of the pneumogastric; also if there is a lesion of the cervical sympathetic (*cf.* p. 23), signs of an aortic aneurism or mediastinal tumour, or if the patient's voice is hoarse. Otherwise it does not form a part of the routine neurological examination.

The pulse, any peculiarities of heart and respiratory functions should be noted here.

An increased pulse-rate without corresponding rise of temperature is indicative of a lesion of the pneumogastric or its nucleus.

On the other hand, a decreased pulse-rate may be the result of vagus irritation (vagotonic conditions), but may also be the result of a lesion of the muscular paths of conduction in the heart (the atrio-ventricular bundle). In complete block of this bundle, I have seen the pulse-rate go down to about 24 a minute (" ventricular rate of contraction ").*

The impressions of taste from the posterior third of the tongue are conducted through the glosso-pharyngeal; this part of the tongue, being rather inaccessible,

* *Cf.* P. F. Holst and Monrad-Krohn: " A Contribution to the Function of the A.-V. Bundle," *Quarterly Journal of Medicine*, 1911, and Monrad-Krohn: *Fasciculus Atrio-Ventricularis*, Oslo, 1911; *Archives des maladies du cœur, des vaisseaux et du sang*, Paris, 1911.

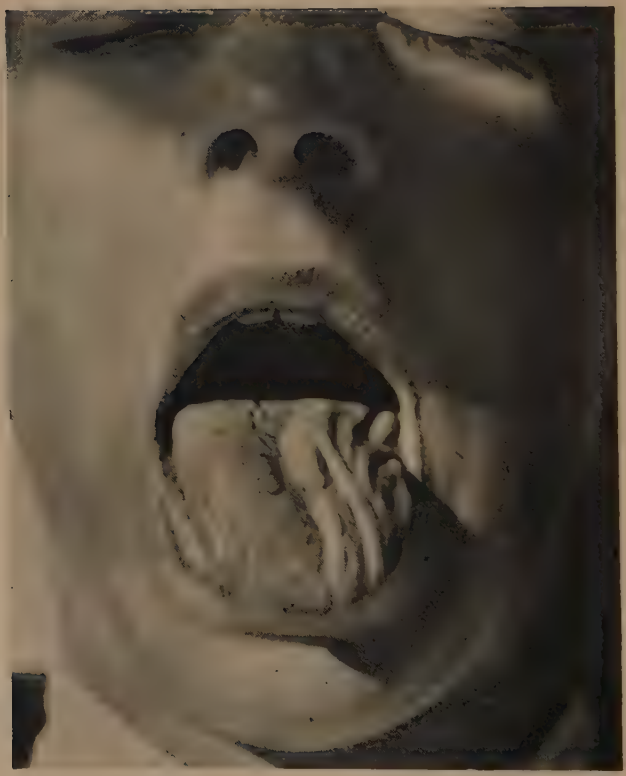

FIG. 22.—PARESIS AND ATROPHY OF LEFT HALF OF THE TONGUE (LESION OF PERIPHERAL MOTOR NEURON).

The tongue deviates to the left on protrusion.

To face page 49.

can only be tested by the galvanic test with a long wire electrode, as described above (under the seventh cranial nerve).

XI. **Accessorius*** (*Motor Nerve for the Sterno-cleido-mastoid and Upper Part of Trapezius*).—The patient is asked to turn his head to the right and to the left; this movement is carried out chiefly by the action of the sterno-cleido-mastoid on the opposite side. The movement is opposed by placing one hand against the patient's chin, while the contracting sterno-mastoid is palpated by the other.

The upper portion of the trapezius is tested by making the patient shrug his shoulders against resistance.

When the upper fibres of the trapezius are paralyzed the scapula alters position, the upper part of the scapula falling away laterally from the vertebral column.

XII. **Hypoglossus** (*Motor Nerve of the Tongue*).—The patient is asked to open his mouth and put out his tongue. Does any *deviation* take place on protrusion ? Is there any atrophy ?

Note if there are any wrinkles on the tongue, and if these are confined to one side. Note if the tongue fills the whole space inside the lower jaw.

In some cases the tongue cannot be protruded at all (*e.g.*, advanced bulbar paralysis, myasthenia) or only for a moment (myasthenia).† If one side is paretic or paralyzed, the tongue will deviate to that side on pro-

* *I.e.*, the spinal portion.

† It has to be kept in mind that an abnormally short frenulum linguæ also may prevent the tongue from being properly protruded. This should, therefore, be inspected.

trusion.* (In cases of facial paralysis the tongue sometimes seems to deviate when it really does not; in such cases the observer must be guided by the relation of the tongue to the teeth;—the interval between the two median incisors corresponds approximately to the middle line.)

The patient is also asked to move his tongue sideways and to press it against the inside of either cheek, while the strength of this pressure is tested with the finger on the outside of the patient's cheek.

Articulation is a complex co-ordinated function involving the co-operation of a number of cranial nerves (fifth, seventh, ninth, tenth, twelfth).

It is most conveniently tested after the examination of the various cranial nerves as just described. If there be a striking peculiarity of the patient's speech, it will probably have made itself manifest during the patient's narrative of the history. In order to detect latent disturbances of articulation the patient is asked to pronounce certain test words: "West Register Street," "British Constitution," "Royal Artillery," "Righteous retribution," "Voluntary contributions," "Hippopotamus," "Irish Constabulary," "Irretrievable."

In cases of dysarthria the attempt at pronouncing these words will in most cases reveal the disturbance.

* This may seem strange to the beginner, who has just been told that in unilateral paralysis of the palate the raphé deviates to the *non-paralyzed side* (*cf.* p. 47). When it is remembered, however, that the lifting of the soft *palate* is due to a *pull*, whilst the protrusion of the tongue is due to a *push*, this difference is easily understood.

In bulbar and pseudo-bulbar paralysis the speech is found to be indistinct; in dementia paralytica the speech is blurred and hesitating, with repetitions and omissions of one or more syllables (the patient stumbles over the syllables—"Silbenstolpern," as the Germans call it); in disseminated sclerosis we find an explosive, staccato speech; and in myasthenia a speech-disturbance which on prolonged effort increases from light dysarthria to complete anarthria.

Palilalia (Souques) is a peculiar disturbance of speech in which the patient repeats a word or a few words in his conversation. These repetitions, which may be very numerous—up to ten or twenty—never occur when the patient reads aloud, recites, or is made to repeat the observer's sentences. It occurs sometimes in post-encephalitic conditions, and is quite different from *stammering* and *stuttering*, which should also be noted and described.

At this stage it is convenient to examine the *cranium* and the *vertebral column*.

Is there any deformity of the cranium, any tumour, any tenderness on palpation or percussion? When the head is strikingly big or small, it has to be decided whether there is macrocephaly or microcephaly. In this respect the measurement of the circumference of the head compared with the measures given on p. 52 will serve as a guide.

In the adult a circumference under 50 centimetres constitutes microcephaly; a circumference over 62 constitutes macrocephaly.*

* As regards X-ray pictures of the skull, see Chapter VII. of the Appendix (p. 201).

Average Circumference of the Head at the Age of—

6 months	..	42·7 centimetres.
12 ,,	..	45·6 ,,
18 ,,	..	46·9 ,,
2 years	..	48·0 ,,
3 ,,	..	48·5 ,,
4 } 5 } ,,	..	50·0 ,,
7 ,,	..	51·0 ,,
10 ,,	..	51·8 ,,

In adults the circumference varies normally—

In men between 53 and 59 centimetres.
In women ,, 51 and 58 ,,

The spine is examined with regard to any *deformity*, *rigidity*, and *tenderness*. When the patient bends forwards, sidewards, and backwards, does the spine curve evenly, or is any portion of it kept rigid? Does any of these movements elicit any pain? Small children may conveniently be lifted by the feet when they are lying face downwards; it is then very easy to see if the spine curves evenly. Is there any tenderness by direct pressure on the processus spinosi, and is there any tenderness on indirect pressure on the top of the head, or on the shoulders, and where is the pain, elicited by such indirect pressure, localized?

Sometimes tenderness is found on direct pressure on the processus spinosi in the lumbar region, but at the same time the tenderness on pressure on the erector trunci is more pronounced; this tenderness is probably in most cases muscular in origin, and (other tests being negative) need give no alarm as regards the condition of the spine.

An X-ray photograph will complete the examination. The spine should always be photographed both in the frontal and sagittal planes.

Tubercular spondylitis, neoplasms, and traumatic lesions of the spine are the chief conditions affecting the spinal cord and the roots. It should be remembered, however, that chronic arthritis of the spine, by causing pressure on the spinal nerves in the deformed and narrowed intervertebral foramina, may give rise to conditions which may in some respects resemble tabes—"pseudo-tabes spondylosique" (Babinski, 1903). Viggo Christiansen has observed a number of cases where this pressure has caused localized pareses.

3. THE MOTOR SYSTEM.

Every movable part of the body is examined with regard to—

(*a*) Posture, shape, and size (any deformity? any *atrophy?*).

Under-development of the bony structures of the limbs is an indication of the causative lesions dating back to the period of growth.

(*b*) Involuntary movements—

Tremor (fine or coarse, influenced by voluntary movements or not).

Fibrillary contractions (twitchings of individual muscle fibres).

Myoclonus (sudden, quick contractions of an individual muscle, usually not producing any movement at the joints).

Athetotic movements (slow, purposeless movements of fingers or toes, taking place

chiefly at the phalango-metacarpal or phalango-metatarsal joints).

Dystonic movements (slow, twisting and turning movements of body and limbs, mostly of large amplitude, "writhings"—*e.g.*, torsion spasm).

Choreatic movements (jerky, irregular, purposeless, constantly changing movements of limbs, body, or face).

Tics (more or less complex movements, always reproduced in the same definite pattern, guided by an obsessional motor impulse which can be voluntarily checked for a time at the expense of an increasing feeling of unrest or malaise. They cease during sleep).

Convulsions (attacks of more or less generalized muscle spasms, tonic or clonic). Their character (tonic or clonic), distribution, and their way of spreading may give valuable diagnostic hints (*cf.* Jacksonian attacks, p. 188).

One should never content oneself with just labelling the involuntary movements seen, but in every case *describe* them and their relation to posture and voluntary movements.

There are involuntary movements which are difficult to fit into any classification.

Especially in post-encephalitic conditions a number of varied involuntary movements are seen, many of which do not correspond to any of the above-mentioned forms and which in former days would be regarded as "hysterical."

For these no adequate collective name can be found apart from the non-descriptive "*post-encephalitic movements.*"

Two particular forms of these, however, should be mentioned—viz.:

(i.) *Involuntary bradykinetic movements* ("Bradikineses," of Pierre Marie and Gabrielle Levy), slow, regular, rhythmical movements of large amplitude at the proximal joints—usually combined, synchronous movements of two limbs. Average rhythm 40 a minute.

(ii.) *Megalo-tremor*, coarse, rhythmical shaking movements, chiefly at the wrist- and elbow-joints in the elevated upper limb. Average rhythm 160 a minute.

(*c*) Passive movements — muscular tonus* (any hypertonus (*rigidity*) or hypotonus? any contracture?† any ankylotic changes in the joints? any pain on passive movements? —if so, this will also prevent active movements from being carried out with full

* *Muscular tonus*, or muscle tone, has been defined in many different ways. For practical clinical purposes it may be defined as the *permanent involuntary tension of the muscle when it has been voluntarily relaxed.* It may vary in intensity according to certain synkinetic and complex reflectory influences. Muscular tonus is generally considered to be composed of two elements: myoplastic or *contractile* tonus depending on cerebro-spinal innervation, and sarcoplastic or *plastic* tonus depending on sympathetic innervation (Langelaan). It has also been postulated that there are two kinds of fibres in the striated muscle: large fibres innervated by medullated (cerebro-spinal) nerves and slender fibres innervated by non-medullated (sympathetic) nerves. The latter would be comparable to the fixing muscles of invertebrates. The importance of the sympathetic innervation in regard to muscle tone seems doubtful according to investigations by Kanavel, Pollock, and Davis. See *Archives of Neurology and Psychiatry*, February, 1925, vol. xiii., No. 2, p. 197, and *Journal of American Medical Association*, November 15, 1925.

† *Cf.* also p. 109.

force; this must not be confounded with paresis *sensu strictiori*).

Besides noticing if the limbs offer any resistance to passive movements (the patient being asked to keep his limbs quite flaccid), it is important to note whether the limbs, after being moved from their original attitude, keep the new position with the same resistance to movements (in either direction), as was observed in the original position.* Such *plasticity* is generally found in the " extrapyramidal " rigidity of the paralysis agitans type,† whilst the rigidity in pyramidal lesions is generally not associated with such plasticity; on the contrary, in pyramidal lesions one often notices a persistent tendency to return to one particular position of predilection.‡ (Thus in the ordinary capsular (pyramidal) hemiplegia flexion at the finger-joints, flexion and pronation at the wrist- and elbow-joints and adduction at the shoulder-joint—in the lower limb extension at all joints are the positions of predilection commonly seen—Wernicke, Mann.)

The *distribution* of hypertonus or hypotonus should

* This plasticity is, according to Foix, due to "postural reflexes" (*cf. La Presse Médicale*, 1924, ii., p. 630), in my opinion, a very unhappy name, which ought to be reserved for the reflexes described on p. 111.

† The "*paradoxical contraction or phenomenon of Westphal*" is seen in this plasticity. It is a contraction which arises when the two points of insertion of a muscle are being approached passively to one another. When thus the foot is pushed upwards, a paradoxical Westphal contraction of the anterior tibial muscle group will fix the foot in this dorsiflexed position.

‡ Here the " postural reflexes of Foix " are lost, whilst the postural reflexes in the sense used on p. 111, on the contrary, are exaggerated, being released from pyramidal control.

always be carefully investigated. The tonus may be quite different in the different muscle groups, and may also vary according to the position of head and limbs (*cf.* postural reflexes, p. 111). When the extensor tonus is stronger than the flexor tonus, an extensor contracture will result and *vice versa*.

(*d*) Active movements—

1. Rapidity of movement (slowness of movement is often the only sign of slight pyramidal lesion).
2. Strength of movement.
3. Range of movement.

Symmetrical parts should always be compared.

In addition to the test for these motor functions pure and simple the following must also be examined:

(*e*) The co-ordination by means of more complicated movements.

These will be dealt with separately after the simple motor functions.

The different parts of the body are best examined in the following order:

1. **Movements of the Head** (partly tested under the Eleventh Cranial Nerve).—Note the characteristic fixation of the head in paralysis agitans and in cervical spondylitis (in the latter the patient often supports his head with the hands).

Is there any retraction of the head? Any resistance to passive movements (on account of pain, meningitis,

torticollis rheumatica, spondylitis, or on account of psychomotor disturbance: negativism) ?

The active movements to be tested consist in bending the head forwards, backwards, and sideways. The rotation of the head is already tested under the eleventh cranial nerve.

2. **The Upper Limbs.**—Atrophy of the thenar, hypothenar, and the intrinsic muscles of the hand (as seen in progressive muscular atrophy, in syringomyelia, lesion of inferior roots of brachial plexus, and lesion of the ulnar nerve—leprosy), must particularly be looked for and noted.

FIG. 23.—PARALYSIS OF RIGHT MUSCULO-SPIRAL NERVE —WRIST-DROP.

The patient is trying to extend both hands.

Lesions of each of the three chief motor nerves of the arm produce characteristic attitudes of the hand.

(i.) In *ulnar paralysis* the two ulnar fingers assume an attitude of hyper-extension at the metacarpo-phalangeal joints and flexion at the interphalangeal joints, due to paralysis and loss of tone of the interossei and the two ulnar lumbricales. The radial lumbricales are only exceptionally innervated from the ulnar nerve (as a rule,

from the median nerve). As a rule, therefore, the attitude just described is only or chiefly seen in the two ulnar fingers. When all the fingers assume this attitude, one may talk of a "claw-hand" ("main en griffe," "Krallen-hand").

The fourth and fifth fingers are also, as a rule, slightly abducted.

(ii.) In *median nerve* lesions we meet with the flat, "simian" hand, the movement of opposition of the thumb being abolished (paralysis of the opponens pollicis).

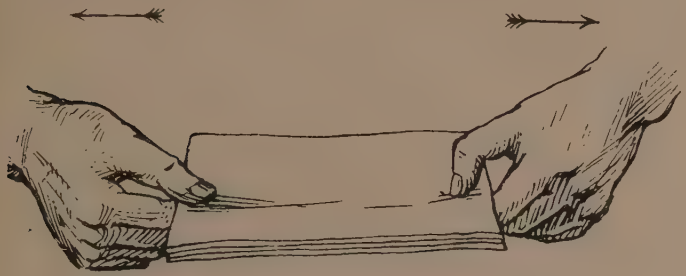

FIG. 24.—FROMENT'S "SIGNE DE JOURNAL" IN LEFT ULNAR PARALYSIS.

Left thumb is flexed at interphalangeal joint—right (normal) thumb is not. Each hand is trying to pull the paper away from the other.

(iii.) In paralysis of the *musculo-spiral nerve* the hand hangs down, unable to be extended dorsally (*wrist-drop*, *cf.* Fig. 23). When in musculo-spiral paralysis the patient grasps anything very firmly, an involuntary flexion at the wrist-joint takes place.

The movements themselves are examined in the following order:

(1) Movements of fingers: flexion and extension, abduction and adduction, the two latter movements tested by making the patient separate his fingers widely,

and then pressing them together again. The examiner can, by putting his own fingers in between those of the patient's, oppose the movement, and thus get an impression of the strength with which it is carried out.

Opposition of the thumb (median nerve). Let the patient put the thumb firmly on the tip of each of the other fingers in succession, and test what power is required for separating them.

For testing the adductor of the thumb (ulnar nerve), the patient is asked to grasp a book or a folded newspaper firmly with both hands and pull the hands away from each other without releasing the grip. When the adductor pollicis is paralyzed the thumb is flexed at the interphalangeal joint during this test, owing to an involuntary contraction of the flexor pollicis longus (Froment's *signe de journal*, *cf.* Fig. 24).

For testing the abduction of the thumb it is well to have the hand resting, palm downwards, on a flat board, a desk, or a book.

(2) The power of the grip of the whole hand. (This can be measured by dynamometer.) It should also be noted if the patient can release the grip at once (if not, this phenomenon is described as *tonic perseveration—myotonic* grip).

Although the grip of the hand is chiefly the result of contractions of muscles innervated from the median and ulnar nerves (chiefly the former), yet the grip is also greatly affected in musculo-spiral paralysis. Owing to the paralysis of the extensors, the fixation at the metacarpo-phalangeal joints is missing; this renders the grip weak and the grasping

of objects clumsy; an attempt at a firm grip is in musculo-spiral paralysis accompanied by a strong, involuntary flexion at the wrist-joint.

(3) Movements at the wrist-joint.

(4) Pronation and supination.

(5) Flexion and extension at the elbow-joint.

(6) Movements at the shoulder-joint—(*a*) movements with extended arms in the frontal and sagittal planes; (*b*) rotation at the shoulder-joint, most conveniently tested with the arm flexed at right angles at the elbow.

(7) Movements of the shoulders—upwards, downwards, forwards, backwards.

In right-handed people the rapidity and strength of movements are generally greater in the right than in the left hand.

In addition to this routine testing of all movements at the different joints, it is often desirable to single out certain muscles for isolated examination. Based on his anatomical knowledge (or aided by his textbook on anatomy), the observer can easily himself improvise the necessary tests. Here only a few muscles which are of particular importance will be mentioned separately.

Pectoralis Major.—Patient presses the hands together, arms extended in front of him; examiner palpates the muscle.

Latissimus Dorsi.—Patient presses his arm downwards against resistance, the arm being extended sideways in the horizontal position; this is chiefly a function of the latissimus dorsi, and the lateral edge of the muscle can

be seen and felt. It is also felt when the patient is asked to cough.

In hysterical paralysis of the arm the latissimus dorsi is sometimes found to be paralytic when tested by downward pressure of the arm, whilst on coughing it is felt to contract quite well.

The same dissociation is also very marked in simulated paralysis of the arm, and is very valuable for diagnostic purposes.*

Serratus Magnus.—Note the position of the scapula when the patient stands with both hands hanging down by the sides. Normally the median border of the scapula is approximately parallel to the spine. In paralysis of the serratus magnus the inferior angle approaches the spine, and in very pronounced cases already in this position one may see a suggestion of winged scapula (*scapula alata*). As a rule, however, this first becomes manifest when the patient tries to keep his arms extended forwards in the horizontal position.

When the paresis is very slight it may be that the "winging" (viz., the lifting off of the median border of the scapula) only occurs when the patient with arms extended forwards presses his hands against the wall, at the same time leaning well forwards, so that the weight is brought on to the arms. For the sake of comparison it is well to let the patient lean on either hand alternately.

Deltoid.—Ask the patient to lift the extended arm

* *Cf.* the author's article, "On the Function of the Latissimus Dorsi Muscle and a Functional Dissociation in Simulated and so-called 'Functional' Paralysis of the Arm," *Acta medica Scandinavica*, vol. lvi., fasc. i., 1922.

outwards to the horizontal position, and test the strength of this movement. When the extended arm is moved upwards 180 degrees in the frontal plane, the first half of the movement (to the horizontal position) is mainly carried out by the deltoid, the second half (from the horizontal position upwards) mainly by the upper fibres of the trapezius aided by the serratus magnus. The trapezius may compensate to a certain extent for a paralyzed deltoid.

Brachio-radialis (*Supinator Longus*).—Ask the patient to bend his arms against resistance, fist closed and thumbs up. The muscle hereunder becomes prominent and can be palpated. (In lead paralysis this muscle as a rule escapes—a point of distinction between lead paralysis and other paralyses of the musculo-spiral nerve.)

3. **The Trunk.**—If the vertebral column has not already been examined, it may conveniently be examined here (*cf.* p. 52).

(*a*) *The Erector Spinæ.*—The patient, lying face downwards, is asked to raise his head and shoulders. The movement is resisted by one hand on the patient's head; with the other, both erectors trunci are palpated; these normally stand out quite distinctly during this movement.

When the patient is standing, he can be asked to pick something up from the floor. When there is a marked weakness of the erector trunci, the patient can only get up again by successively placing his hands on his knees and thighs, and by means of his arms *pushing* himself up into the erect position (and as a rule beyond the erect position, as these patients generally develop an

habitual lordosis). In pronounced weakness he does not dare to stoop, but bends his knees *ad maximum*, with an erect trunk, in order to pick a thing off the floor (*e.g.*, myopathy).

(*b*) *The Abdominal Muscles.*—Ask the patient (lying on his back) to raise his head from the pillow with and without resistance; note if there is any deviation of the umbilicus, this being drawn away from a paralyzed portion of the muscular abdominal wall.

Ask the patient to cough. A paralyzed part of the abdominal wall bulges on coughing. During this—as also during respiration and whilst the patient is speaking—any deviation of the median line ought also to be looked for.

Can the patient rise from the lying to the sitting position without supporting himself on his arms? This depends chiefly upon the strength—

(1) Of the abdominal muscles; and

(2) Of the ilio-psoas.

Thus, only when the ilio-psoas is good does inability to sit up indicate a weakness of the abdominal muscles.

For this test the examiner, if necessary, holds the patient's feet down, as in this connection it is only the strength of the muscles which is to be tested. When the patient tries to rise without having his feet fixed, it will often be noticed in cerebellar disease that the legs are raised instead of the trunk; in ordinary capsular hemiparesis the leg on the hemiparetic side is raised on attempting to sit up; in hysterical hemiplegia, as a rule, *vice versa*. Normally the legs and feet should remain unmoved when the patient sits up, unless the patient be particularly stout or badly proportioned.

The abdominal reflexes also give some information about the state of the abdominal muscles. In partial paralysis of the abdominal wall a heterosegmental abdominal reflex is found (*cf.* later, p. 104).

(*c*) *Diaphragm.*—Patient is told to perform abdominal respiration. Look at the epigastrium. Does it bulge, as normally it should, during respiration, or is it drawn in during inspiration? The latter points to paralysis of the diaphragm.

(*d*) *The Intercostal Muscles.*—Note the intercostal spaces during inspiration and expiration. In paralysis there is an alternating drawing in and bulging, according to the different phases of respiration, difficult to observe in stout people.

4. **The Lower Limbs.**—Note any deformity of the foot (Friedreich's disease, poliomyelitis). Note any peculiar posture. A pronounced flexion contracture is indicative of a severe spinal cord lesion (*cf.* p. 109).

When the muscles are hypotonic the shape of the thigh is altered in such a way that on section it forms a lying oval **O** ("Breites Bein" of the Germans) instead of a standing oval **O**, which represents the normal section of the thigh.

The movements themselves are examined in order of the joints, thus:

(*a*) Movements at the toe-joints.
(*b*) Movements at the ankle-joints.
(*c*) Movements at the knee-joints.
(*d*) Movements at the hip-joints.

Normally it is not possible to bend the extended leg of an adult against his voluntary resistance. Special indications how to test every single movement seem superfluous; it may easily be gathered from what has already been mentioned. The movements must be tested with

regard to rapidity and range as well as with regard to power (as already pointed out). *All* the movements should be carefully tested; the dorsiflexion at the ankle-joint, the flexion at the knee-joint, and the flexion at the hip-joint are of particular interest, as they are perhaps more frequently affected than the other movements. It will generally be found impossible to prevent a strong adult person from lifting his knee (ilio-psoas). The beginner is often apt to forget the movements of ab- and adduction at the hip-joint; these ought to be tested both with extended and flexed legs.

Kernig's sign will have been detected whilst testing the passive movements. This sign is said to be

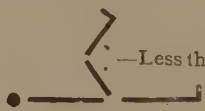

FIG. 25.

present when the angle between the calf and the thigh in maximal flexion cannot be opened to 1½ right angles.

The other leg must be kept down and fully extended. This sign (as retraction of the head) points to a meningeal irritation.

For quite young children Kernig's sign is unreliable. For many cases of meningitis during the first two years it is missing, and, on the other hand, it is also found in other conditions at this young age.

At the same time *Lasègue's* sign of sciatica* (pain along

* Besides the sign here mentioned, there is another phenomenon which is also called *Lasègue's sign*—viz., a patient's inability to move a limb, which he is prevented from seeing.

the sciatic nerve on bending the extended leg at the hip-joint) will also be tested for.

This forms a strong contrast to the tabetic hypotonia, which allows an increased range of all passive movements without any pain being elicited. This contrast is of practical importance, as it helps to distinguish sciatica from tabes; both may cause similar spontaneous pains along the back of the lower limbs, and the so-called "formes frustes" of tabes (where many of the other diagnostic signs of the disease are in abeyance) are not at all rare.

In the differential diagnosis between sciatica and arthritis of the hip-joint the test described by *Patrick* is very useful: With the patient supine in bed the thigh is flexed and the ankle placed on the opposite extended leg as high up as possible, preferably above the patella. The knee is now pressed outwards and downwards, thus causing a combined movement of abduction and outward rotation at the hip-joint. In hip disease pain is elicited before the knee reaches the level of the bed.

Also in *meralgia paræsthetica* (affection of the lateral cutaneous nerve of the thigh), I have found that the Patrick test may elicit pain; but in these cases pure abduction at the hip-joint rarely causes any pain.

In connection with Kernig's sign the *Brudzinski sign* ought to be looked for. The head of the patient, who is lying flat on his back, is passively flexed. If this determines a flexion at the hip- and knee-joints the sign is said to be present.

The *sign of Guillain* should also be looked for in this connection. It consists in a brisk flexion at the hip- and knee-joints, elicited by means of pinching the contralateral quadriceps muscle. Both these signs are indicative of meningeal irritation.

Once more it has to be emphasized that, whenever

possible, symmetrical parts should always be compared.

Just as there is a sensory segmentation of the skin (see later), there is also a motor segmentation of the muscular system; the spinal segmentation of some of the principal muscles follows:

C_4 =diaphragm.
C_5 =deltoid.
C_6 =biceps, brachio-radialis, pronators.
C_7 =triceps.
C_8 =interossei, long flexors of fingers.
D_1 =muscles of thenar and hypothenar.
D_{2-12}=intercostals.
L_{1-2} =ilio-psoas.
L_3 =quadriceps.
L_4 =adductors.
L_5 =peronei and "peroneal group."
S_1 =gastrocnemii, hamstrings.
S_2 =glutæi, intrinsic muscles of foot.
S_{3-5} =muscles of perineum.

Associated Movements.

Synkinetic Movements.—Certain voluntary movements have a tendency to be accompanied by other, involuntary movements, which are called "associated" or "synkinetic" movements (*Mitbewegungen*). Such movements exist normally—*e.g.*, the pendular movements of the arm accompanying the gait, facial contractions on violent exertion In pathologic conditions such associated movements may be lost or exaggerated or new synkinetic movements may appear. In "extrapyramidal" motor lesions of the paralysis agitans type there is a marked diminution or loss of all associated movements (*Bewe-*

gungsarmut). In pyramidal lesions, on the other hand, we find a number of associated movements, many of which are not encountered in the normal individual. These associated movements may have such a disturbing influence on the voluntary movements, which they accompany, as to cause a serious motor impediment.

For the sake of an easy survey we may arrange these associated movements in three classes:

1. The generalized synkinetic movements of the ordinary pyramidal hemiplegia ("syncinesie globale ou spasmodique").

2. The symmetrical associated movements ("syncinesies d'imitation").

3. "Co-ordinated" associated movements ("syncinesies de coordination").

1. *The generalized associated movements of pyramidal hemiplegia* consist in movements tending to produce the typical attitude of predilection (contracture)—*i.e.*:

(*a*) In the upper limb: flexion of fingers, flexion and pronation at wrist- and elbow-joints, adduction or abduction at the shoulder-joint.
(*b*) In lower limbs: extension at all joints.

These synkinetic movements in pyramidal hemiplegia are readily elicited by all kinds of exertion. Thus they often appear when the patient is made to squeeze the observer's hand as hard as he can with his non-paralyzed hand. During this associated facial movements may also be seen to be exaggerated in the paretic side of the face (*cf.* Figs. 17 and 18).

2. *The symmetrical associated movements* consist in involuntary movements of imitation in the paretic limbs accompanying voluntary movements of the healthy limbs on the other side. They appear particularly when the patient has to exert himself, carrying out quick or strenuous movements.

3. *The "co-ordinated" associated movements* consist in involuntary movements of synergic muscle-groups, accom-

panying a voluntary movement in a paretic limb. The Strümpell phenomenon and the finger sign of Souques are typical instances of such " co-ordinated " associated movements (see below).

A number of these associated movements are of great practical value, particularly for the distinction of pyramidal from hysterical paralyses. Of a great number of synkinetic movements, the following are some of the most important:

The *combined flexion of the hip and trunk:* The patient lies flat on his back, both arms crossed over the chest; he is then asked to sit up, and in case of pyramidal hemiplegia the paretic leg is involuntarily lifted up.

The *Strümpell* phenomenon: On voluntary flexion at the knee-joint an involuntary dorsiflexion and supination of the foot occurs.

The *pronator* sign consists in involuntary pronation of the paretic forearm when both arms are extended upwards.

The Finger Sign of Souques.—The patient is asked to lift both arms. In paresis of pyramidal origin the fingers of the paretic hand are extended and separated during this.

Hoover's Sign.—When the patient lying on his back lifts one leg, normally a downward pressure of the other leg is noted (the observer keeps his hand under the heel). This is accentuated in organic central lesions on attempt to lift the paretic leg, but abolished in hysteria.

Observations in my clinic by Dr. Saethre, have shown that in pyramidal hemiplegia a synkinetic movement of abduction at the hip-joint often accompanies flexion at hip- and knee-joints. This is best seen when the patient is standing, and then asked to " mark time." This involuntary abduction at the hip-joint is probably one of the chief factors responsible for the " circumduction " seen in hemiplegia (*cf.* chapter on the Gait, p. 120).

Many of these associated movements may be regarded as reflex phenomena, closely allied to the postural reflexes (*cf.* p. 111).

On the other hand some associated movements, normally present, may be abolished in pyramidal lesions.

The Platysma Sign (*Babinski*).—The patient is asked to open his mouth as wide as he can. This normally entails a contraction of the platysma, which is lost in pyramidal hemiplegia, but present in hysterical hemiplegia.

The Forearm Sign (*Leri*).—When the observer passively flexes the patient's fingers and wrist, normally an involuntary flexion occurs at the elbow. In hysteria this reaction is present as in the normal individual, but in pyramidal paralysis this reaction is abolished.

The Phalangeal Sign (*Mayer, Innsbrück*).—When the proximal phalanges of the second and third fingers are passively flexed *ad maximum*, an involuntary extension and adduction of the thumb will normally take place. In pyramidal lesions this associated movement of the thumb is lost.

Other normal associated movements disappear in extrapyramidal disturbances of the paralysis agitans type—*e.g.*, when a patient sitting in a chair is suddenly thrown back, normally an involuntary extension at both knee-joints occurs. In paralysis agitans this associated movement is often missing (*Souques' leg sign*).*

Co-ordination (*Disturbance: Inco-ordination, Ataxia, Asynergia*).—1. The patient is asked to put his forefinger up to the tip of his nose with his eyes closed.

Does the finger readily find the tip of the nose? Is the movement accompanied by tremor? Does the tremor show the "crescendo" characteristic of intention tremor? Does movement stop a tremor, which is present at rest?

The finger-nose test should be carried out with

* Disappearance of the associated arm movements accompanying the normal gait is a frequent finding both in pyramidal and extrapyramidal lesions.

different speed (first quickly, then slowly). The patient should also be asked to perform the movement in different planes.

Let the patient finally repeat the movement with his eyes open. Does this make any difference ?

2. The patient is asked to lift a glass filled with water to his lips.

3. To button and unbutton his coat (eyes closed).

4. To write his name.

5. He is asked to put the one heel on the other knee, and *vice versa*, with his eyes closed.

6. To write the figure of 8 in the air with his foot (eyes closed).

7. Romberg's test. Ask the patient to stand with his feet close together and eyes shut. Does he just begin to sway ? Must he move his feet in order to keep his balance ? Does he actually fall if not supported ?

The last test is, however, most conveniently put off and examined in connection with the standing position and the gait at the end of the whole examination (see later).

Special Examination for Cerebellar Signs.

1. Cerebellar *hypotonus* will already have been detected by the examination of passive movements. By means of direct palpation of the flabby muscles in repose the hypotonus is appreciated as a rule even better than by passive movements.

2. *Asthenia and slowness* in muscular contractions will have been discovered during the examination of active movements.

3. *Babinski's Tests for Asynergia* ("*Décomposition des Mouvements*").—(*a*) As regards the upper limbs, the asynergia will already have shown itself during the forefinger-nose test. The movement is in cerebellar lesion "decomposed"—*i.e.*, its various components are carried out successively instead of synchronously. The patient may be made to point at various things, always arranging the test so that the intended movement consists of a synchronous combination of two or more simple movements.

(*b*) To test the lower limbs, the patient is asked, when he is *lying* down, to move the foot as near to his seat as possible. Cerebellar "decomposition" will then show the following series of movements:

(1) Flexion at hip-joint; followed by
(2) Flexion at knee-joint; again followed by
(3) Extension at hip-joint.

When the patient is *seated* on a chair he is asked to touch the examiner's hand with his big toe. The hand should be held about 60 centimetres above the floor and an equal distance from the chair. Cerebellar disturbance will show similar "decomposition" as just described.

When the patient is *standing*, ask him to put the foot on the seat of a chair—"decomposition" will again betray itself in the same way.

(*c*) To test the trunk, the patient is asked, when standing, to bend his head and trunk backwards, eyes closed. A cerebellar patient will be incapable of combining this movement with other simultaneous

movements (dorsiflexion at ankle-joint and flexion at knee-joint, or extension at hip-joint) necessary for the maintenance of equilibrium.

When the patient is lying down, ask him to cross his arms over the chest and sit up. Normally a highly complicated movement is carried out, reducing the momentum of the trunk, by shortening the lever upon which the weight is acting, as compared with the lower limbs. Only thus is it possible to sit up normally without lifting the feet. In cerebellar disturbance this highly complicated movement is rendered impossible, and instead of the patient sitting up he will lift both lower limbs. Only when these are kept down by the examiner is the patient able to sit up.

4. *Dysmetria.*—Failure to stop intended movements in time (due to failure [? hypotonus] of antagonists).

The forefinger-nose test, which uncovers such a multitude of disturbances, as a rule also discloses the presence of dysmetria; the finger shoots past the nose on to the cheek, or in an attempt at correction, almost invariably resulting in over-correction, the movement is arrested before it reaches its destination.*

The patient may also be asked to draw a line with a pencil on a piece of paper from one point to another; as a rule a cerebellar patient fails to stop at the second point: on a second attempt he generally stops before he reaches it (over-correction).

5. *Test for the Rebound Phenomenon of Stewart and Holmes.*—The patient supports his elbows on a bed or

* Söderbergh has described this under the name of "le signe de frein."

a table, and is asked to pull each hand in succession towards his shoulder against resistance, the examiner grasping his wrists. When the resistance is suddenly released, the hand in cerebellar lesion flies up to his mouth or shoulder, often with considerable violence, whilst the movement of the normal limb is arrested almost immediately by contraction of the antagonists (triceps), and may even be jerked back or rebound.

(In spastic limbs the rebound may be excessive.)

6. *Test for Dysdiadochokinesis or Adiadochokinesis (Babinski).*—Viz., inability to execute alternating opposite movements as quickly and regularly as a normal person.

The patient is asked to pronate and supinate either forearm in the quickest possible succession. In cerebellar lesion the movements are much slower and more irregular than normal, often with a distinct interval between each pronation and supination.

Very often in right-handed persons a slowness on the left side as compared with the right is found.

In a limb, affected with spastic paresis due to a pyramidal lesion, dysdiadochokinesis is invariably found, and has then no value as a cerebellar sign.*

7. *Spontaneous Deviation.*—The patient holds both arms extended horizontally in front, and then closes his eyes. In most cases of unilateral cerebellar lesion, the

* Theoretically there should be a difference inasmuch as in cerebellar lesions the movements of pro- and supination in themselves are not retarded (as in spastic paresis); only prolongation of the interval between the two antagonistic movements should be responsible for the cerebellar dysdiadochokinesis.

homolateral arm swings away from the symmetrical position. This spontaneous deviation is also elicited by—

8. *Barany's Pointing Tests.*—The patient is asked to touch the examiner's forefinger with his own. He is then asked to close his eyes and to drop his hand and arm, and again try to bring it back to the original position touching the examiner's finger, which must be kept quite still.

The test of moving the finger away from and back to the examiner's finger as a target, with eyes shut, is tried in both the horizontal and the vertical planes and for each joint separately. Only a constant deviation ("past-pointing") in one and the same direction is counted.

The various vestibular tests may also throw light on a cerebellar disturbance (*cf.* pp. 42 and 73 and Appendix IV., p. 173).

9. Finally, the patient is told to walk along a straight line with his eyes shut. In unilateral cerebellar lesion there is a tendency to homolateral deviation.

If the patient cannot walk without support, it is advisable to make him crawl on the floor with his eyes shut. This in unilateral cerebellar lesion often reveals a tendency to roll over to the side of the lesion.

10. The patient should also be made to walk forwards and backwards (six to seven steps in either direction is sufficient) with his eyes shut (or blindfolded).

The deviation in cerebellar affection will then often show in a peculiar way. The patient will gradually turn round and the tracings of his path will roughly resemble a mariner's compass (*cf.* Fig. 26)—"compass gait."

These walking and crawling tests may conveniently be carried out in connection with the routine inspection of the gait at the end of the whole examination.

All the above-mentioned cerebellar signs are homolateral, in contradistinction to most cerebral phenomena.

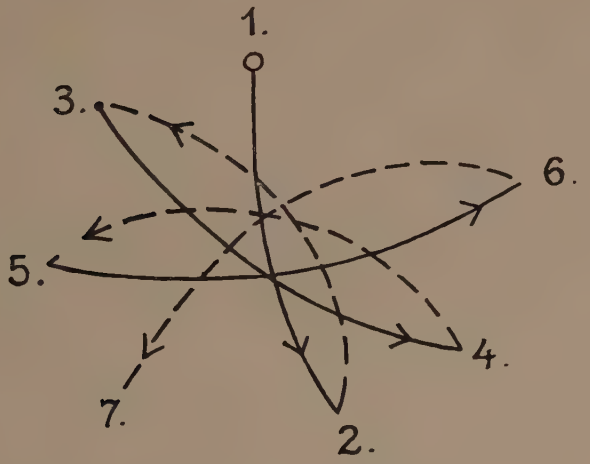

FIG. 26.—"COMPASS GAIT."

1 signifies the starting point; the *continued* lines indicate the tracing of the patient's *forward* walking; the interrupted lines his backward walking. (Monrad-Krohn: "A Case of Fibrous Meningioma in the Cerebello-Pontine Angle," *Acta Psychiatrica et Neurologica*, vol. iv., fasc. 3, 1929.)

The cerebellar localization is not so well known as the cerebral one. Yet we are probably justified in assuming that lesions in the *superior, anterior parts* of the cerebellar hemispheres cause disturbances of co-ordination in the homolateral *upper limb*, whilst lesions in the *inferior, posterior* parts of the cerebellar hemisphere cause disturbances of co-ordination in the homolateral *lower* limb (Ingvar)

Barany assumes centres of "directional tonus" (upward-, inward-, downward-, and outward-tonus) for the different limbs in the cerebellar hemispheres. Destruction of any of these "directional" centres should cause spontaneous deviation of movements in the opposite direction (*e.g.*, by destruction of an outward-tonus centre past-pointing inwards and *vice versa*.)

4. SENSORY SYSTEM.

The sensory examination is no doubt the most difficult part of the whole examination. Care must be taken not to tire the patient out, as a tired patient is generally a very bad witness. It is better to aim at a quick orientation regarding the *whole* sensory system during the first examination, and not at first bother too much about details, which can easily be elicited at a subsequent examination—*e.g.*, the next day, when the patient is rested.

During the whole sensory examination the patient ought to have his eyes closed or covered. Great pains must be taken to avoid exerting any suggestion. Consequently, he must not be asked, "Do you feel that?" each time he is touched; but simply be asked to say "Yes" every time he feels that he has been touched,* "Oh" every time he feels pain, "Warm" every time he has a sensation of warmth, and "Cold" every time he has a sensation of cold.

When in this way the whole body has been overhauled, the examination may be completed by asking the patient

* In simulated anæsthesia an unintelligent or flustered patient will sometimes say "No" on being touched.

to compare the stimuli applied to different parts of the body.

A great deal of attention on the part of the patient is required, and this had better be impressed upon him at the outset of the examination.

The interval between the different stimuli ought to be varied, as experience shows that the attention of the patient is often lulled to sleep by rhythmical stimulation. The patient must be urged to answer *as soon* as he has a sensation, as otherwise the phenomenon of *delayed sensation* may be overlooked.

In recording the sensory changes the examiner ought always to indicate exactly *in regard to what stimulus* he has found *anæsthesia* (complete loss of sensation), *hypoæsthesia* (diminished sensation), or *hyperæsthesia* (increased sensation).

It is convenient to mark the areas of sensory changes with a skin-pencil, and at the end of the examination transfer the findings to one of the usual sensory charts.

The different forms of sensation are examined in the following order:

I. Superficial Sensation.

(A) *Tactile sensation, elicited by light touch with a fine, soft paint-brush, a wisp of cotton-wool, or, best of all, a piece of paper.*

By always using a piece of paper of the same kind, and the same size and shape, and by always holding it in the same position when touching the patient, the stimulus will

practically always have the same strength. A piece of paper is therefore nearly as good as an æsthesiometer. It has to be kept in mind also that the result of the sensory examination depends on so many different factors (fatigue, attention) of a purely subjective order that it can never be rendered quite objective. This impairs the value of any æsthesiometer.

(B) *Superficial algesia* (*i.e., sensation of pain*) *elicited by pinprick.* In order to investigate the *sense of pain,* the examiner should prick hard enough to cause an *unpleasant sensation* (this is easily tested on his own hand); to ask the patient to distinguish between touch with the point and the head of the pin is no proper test for cutaneous pain.

The examination by means of pinprick is the easiest and quickest way to attain a rough orientation of the state of the patient's superficial sensation, and if the patient is a little tired, or if his attention is beginning to flag, the examiner will do well to confine himself to the pinprick test, and postpone the test of the other qualities of superficial sensation to the next day.

Areas of superficial *hyperæsthesia* are easily detected by stroking the skin with a pin. The moment an hyperæsthetic area is touched the patient winces. The determinations of hyperæsthetic areas are of great value, as they occur partly as stigmata in hysteria, partly as the result of a " viscero-cutaneous reflex " in lesions of the various internal organs (Dana, Head, and others).

(C) The sense of *temperature* is examined by means

of *test-tubes*, preferably of very large size, filled with cold water from the tap, and hot water (about 50° C.).

The sensitiveness to heat and the sensitiveness to cold is very different in the different cutaneous regions, and not quite parallel to one another. Thus the trunk and the proximal parts of the limbs are generally more sensitive to cold than the distal parts of the limbs are (the uncovered parts of the body less sensitive to cold than the covered parts of the body). For *exact* comparisons, therefore, only symmetrical areas can be used, and then only when tested under identical conditions. (Thus one should not compare a region just uncovered with one that has been uncovered during the whole time of the examination.)

Whenever possible, it is well to ascertain the minimum differences of temperature which the patient can perceive in the various parts of his body.

For the sake of a first rough orientation it will be sufficient to test the sense of temperature by means of the electrical hand-lamp, kept at a distance of 5 centimetres from the skin.

(D) The *sense of discrimination* is tested by simultaneous touch with the two points of Weber's compass or Holmes's compass or a carpenter's calipers; the patient is instructed to say "One" or "Two," according to whether he feels the touch of one or two points. The minimum distance between the two points, when felt separately, is noted. Normally this distance is largest in the direction of the longitudinal axis of the limbs, and smallest in the direction perpendicular to this.

The distance varies considerably in the various parts of the body—*e.g.*:

Finger-tips	3 to 8 mm.
Palm of the hand	8 to 12 ,,
Back of the hand	30 ,,
Chest, forearm, shin	40 ,,
Back	40 to 70 ,,
Upper arm, thigh	75 ,,

(E) *Discrimination of Touch with Different Materials.*—For this purpose swabs of cotton-wool, covered with materials of different texture (silk, emery cloth, wool, linen, etc.), are tied on to the blunt ends of ordinary pencils and the patient invited to tell the difference when touched or stroked with them in succession.

(F) *Localization.*—The patient is asked to indicate the exact situation of each sensation he has; this is most conveniently examined in connection with (A) and (B).

Head has created the distinction between *epicritic* and *protopathic* sensibility, the first comprising light touch, small variations of temperature, and discrimination of two points; the latter comprising pain and extremes of heat and cold.

In a peripheral nerve lesion the epicritic disturbance is more extensive than the protopathic; but the nearer the lesion to the central nervous system, the smaller this difference, and in posterior root lesions the area of analgesia to pinprick (protopathic sensibility) exceeds that of anæsthesia to light touch (epicritic sensibility).*

Electrical stimulation is sometimes used in the ex-

* "C. H. Frazier and S. Silbert have come to the conclusion that the extensive experience on nerve lesions gained in the War has given a death-blow to Head's theories of sensation, and to the distinction between epicritic and protopathic sensibility (*Practical Medicine Series*, Chicago, viii., 1920, p. 138).

amination of the sensory system. According to Le Dantec, the different forms of electrical stimulation correspond to the different forms of superficial sensation,

Faradic sensation corresponding to the sense of touch,*
Galvanic sensation corresponding to the sense of temperature,
Sensation of electric sparks corresponding to the sense of pain elicited by pinprick.

The electrical examination of sensation is of little practical value.

II. **Deep Sensation.**

(A) *"Joint Sense" (Sense of Position, Sense of Movement).*—The different parts of the patient's limbs are moved, and he is asked to tell in what position the limbs have been placed. He must, of course, keep his eyes shut and his limbs as flaccid as possible, avoiding all voluntary movement. While moving the patient's limbs, the examiner must aim at exerting as uniform a pressure as possible with his hands, so as to give the patient no information of the movement through the superficial sensation.

One can also try to find the threshold of the sense of movement at the different joints, asking the patient to say "Yes" as soon as he feels movement at a particular joint. Then he is asked in what direction the movement has taken place. The normal threshold value is about or below 1 degree for most of the joints; for the ankle-joints and the interphalangeal joints it is between 1 and 2 degrees.

* According to Frazier and Silbert loss of faradic sensation is fairly good evidence of complete interruption in peripheral nerve lesions.

Each joint has to be examined separately.* The big-toe joint (metatarso-phalangeal) is perhaps the one which is most commonly affected (*e.g.*, in tabes).

As regards the hands and the fingers, it is a very good plan to ask the patient to close his eyes, and tell him to put the fingers of the one hand in the same position as the examiner places the fingers of the other.

The patient may be asked to hold his hands in front of him, palms down, fingers separated, arms extended; then to shut his eyes and keep the hands quite still. In case of loss of joint sense the fingers, hands, or arms will move slowly away from the original position. As regards the fingers, this has been called "tabetic athetosis," on account of some external likeness in the movement, but this phenomenon has a widely different pathogenesis from the real athetosis, which is probably a mid-brain symptom.

The joint sense is more often affected in the small joints of the distal parts of the limbs than in the large proximal joints.

A valuable complex test which comprises a number of joints is the following *Finger heel-pointing test.*† The patient, lying flat on his back with his eyes shut, is asked to point his forefinger at his heel, which latter the observer moves about, carrying out various passive movements at the knee- and hip-joints. First make him point the right forefinger at his left heel, then his left forefinger at the right heel.

* The author often employs an apparatus which registers the threshold of perception of movements at the hip- and knee-joints. The apparatus cannot be claimed to secure absolutely exact data, but it may be found of some help in *recording* the amount of loss of joint sense. It has been described in *Norsk Magazin for Lægevidenskaken*, 1915.

† This complex test, devised by the late Dr. Saunders, will generally unmask even a very slight loss of joint sense in the joints involved in the test.

(B) *Deep Pressure Pain.*—Pinch the muscles of the forearm, overarm, thigh, calf, and the tendo Achillis,* and note what amount of pressure is required to elicit pain. Also press on the eyeballs, larynx, the epigastrium, and the testes (normally these parts are very tender to pressure). An algometer may be employed, and renders an approximate quantitative test possible.

Deep pressure pain is frequently in abeyance in tabes and tabo-paresis; on the other hand, it is frequently exaggerated in neuritis, where the deep hyperæsthesia is often excessive.

Also the nerve trunks ought to be tested by pressure or gentle percussion. In case of peripheral nerve injuries pressure on the nerve will, if regenerating neuraxes have reached the point pressed, elicit a tingling sensation in the peripheral distribution of the nerve. This sign—*Tinel's sign*—can thus give information regarding the process of regeneration.

The nerves should also be explored by means of *palpation* in order to detect any *swelling*.

The chief nerves to be explored by means of palpation are the following: the ulnar nerve, the great auricular nerve, the superficial peroneal nerve, and the supra-orbital and frontal nerves.

For palpation of *the ulnar* nerve the observer should sit in front of the patient and palpate with the hand which is diagonally opposite the arm whose ulnar nerve is to be examined. With the tips of the second and third fingers the nerve is pressed towards the

* Abadie's sign: absence of pressure pain in the tendo Achillis frequent in tabes.

epicondylus of the humerus. As the nerve finally slips lateralwards under the finger, a fair impression of its thickness is obtained. The inexperienced must be warned that not every ulnar nerve easily felt is pathologically thickened. It requires great experience in palpating the normal nerve to decide whether it is pathologically thickened or not. For purposes of comparison one cannot be too careful in always palpating accurately in the same way and in the same position.

The *great auricular* nerve may be easily felt where it passes across the sternomastoid muscle. One finds the nerve crossing this muscle at a distance of 1 to 3 centimetres behind the external jugular vein, and running roughly parallel to this vein. Where the external jugular vein is not visible, it can, as a rule, be made visible by slight pressure with the hand at the side of the neck. This will make the vein bulge and betray itself.

The *supraorbital* nerves (and the *frontal* nerves) are best palpated when the examiner stands behind the patient, who is sitting. Both hands can then be simultaneously employed in palpating the supraorbital edge and adjacent parts of orbita and the forehead—on both sides.

In *leprosy* many of the peripheral nerves may be distinctly swollen, and, in the more acute stages, also tender. The swelling may be even or nodular.

In *hypertrophic forms of chronic polyneuritis* (Déjerine, Sotta) the swelling of the nerves may also be ascertained by means of palpation.

(C) *Vibration.*—A large tuning-fork is placed on the styloid processes of radius and ulna, on olecranon, claviculæ, sternum, crista ilii, patella, malleoli, and the patient is asked if he feels the vibration. It has to be

made quite clear to the patient that it is the sensation of vibration, not the touch nor the sound, to which he has to direct his attention. In tabes the test applied to the processus spinosi often gives an instructive segmental diagnosis.

III. **Combined Sensation.**

Stereognostic Sense (loss of stereognostic sense = astereognosis).—The patient is asked to close his eyes, then various familiar objects are put into either hand, and he is asked to name them; or, failing this, to describe them (size, shape, material). (N.B.—Do not use a bunch of keys; the sound produced by the rattling of the keys will tell the patient what it is.)

This test is of great practical interest, *e.g.*, in hemiplegia; if a hand remains astereognostic it is practically useless, even if it regains its motor power completely.

The test of stereognostic sense, as just described, is really a test of perception as well as sensation, as it requires a certain amount of associative elaboration. To name the different things is a psychosomatic function, and this test is also made use of in the complete psychosomatic examination (see later). In this connection it is sufficient for the patient to be able to give some simple description of the object felt as to size, shape, and material.

Sensory Paths and Segmentation.

The fibres conducting *superficial sensation* (pain, temperature, and partly touch) decussate immediately or very soon after their entrance into the spinal cord, and ascend in the antero-lateral columns through the

spinal cord and the brain-stem to the optic thalamus. In their decussation in the cord these fibres come into close relation to the central canal. (N.B.—Syringo-myelia.)

The fibres conducting *deep sensation* ascend uncrossed through the posterior column to the nuclei of the fasciculi of Burdach and Goll; the fibres decussate above these nuclei (*decussatio lemniscorum medullæ oblongatæ*) and ascend to the optic thalamus, joining the fibres for superficial sensation (*cf.* Figs. 50 and 51 at the end of the book).

It is easily seen how the different anatomical relations of the different paths of sensation in the spinal cord may cause various kinds of *dissociation of sensory loss* as the result of limited lesions.*

Some Landmarks for the Segmental Diagnosis.—The surface of the body can in respect of sensory innervation be divided into segments, the distribution of which is easy to remember, when it is kept in mind that we were originally quadrupeds (*cf.* Fig. 27, p. 90).

The *ulnar border of the upper limb* corresponds to first and second dorsal segment (often hypoalgesic in tabes).

Papilla mammæ corresponds to the fifth dorsal segment.

Umbilicus corresponds to the ninth dorsal segment.

Planta pedis corresponds to the first sacral segment.

The front of the leg corresponds to the lumbar segments; the back of the leg to the sacral segments.

The *perianal area* corresponds to the fourth and fifth sacral segments. The segmental areas are subject to individual variations (*cf.* also Figs. 54 and 55).

* It has to be remembered, however, that also quite peripherally the elements of the various forms of sensation are anatomically separated from one another (*cf.* the heat, cold, and pain spots of the skin).

It has to be remembered that the different spinal segments are not situated at the same level as the numerically corresponding vertebræ (*cf.* Table below).

TABLE SHOWING APPROXIMATE RELATIONS OF SPINAL SEGMENTS TO SPINOUS PROCESSES.

Spinal Segment.		*Spinous Process.*
$C_{1\ 2}$	above corresponds to	C_1
C_3	" "	C_1
C_4	" "	C_2
C_5	" "	C_3
C_6	" "	C_4
C_7	" "	C_5
C_8	" "	C_6
D_1	" "	C_7
D_2	" "	D_1
D_3	" "	D_2
D_4	" "	D_{2-3}
D_5	" "	D_{3-4}
D_6	" "	D_{4-5}
D_7	" "	D_{5-6}
D_8	" "	D_6
D_9	" "	D_7
D_{10}	" "	D_8
D_{11}	" "	D_9
D_{12}	" "	D_{10}
L_1	" "	D_{10-11}
L_2	" "	D_{11}
L_3	" "	D_{11-12}
L_{4-5}, S_1	" "	D_{12}
S_{2-5}	" "	L_1

The lower end of the spinal cord is in the adult at the level of the first or second lumbar vertebra (in the new-born, the third lumbar vertebra).

As a rule, the upper border of the sensory loss in medullary lesions corresponds to the level of the lesion. Yet it must be remembered that spinal tumours at

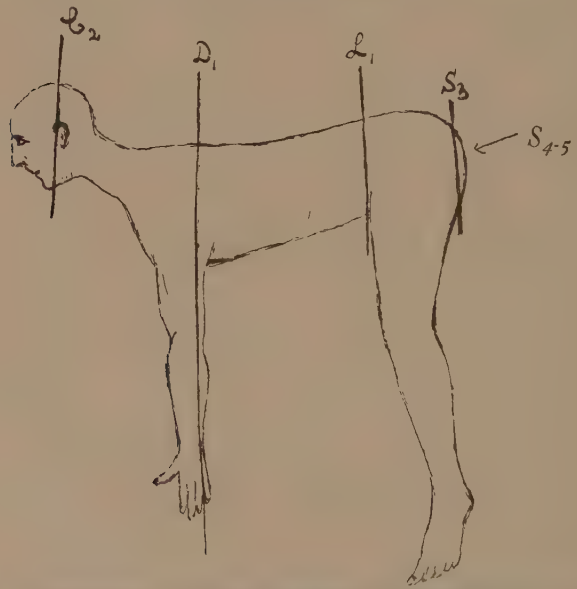

FIG. 27.—MNEMONIC DIAGRAM OF SENSORY SEGMENTATION (ONLY APPROXIMATE TOPOGRAPHIC EXACTNESS, CONSIDERABLE INDIVIDUAL VARIATIONS).

first may produce a sensory loss the upper border of which may be situated *considerably below* the level of the lesion. This has in several cases led to diagnostic errors (Elsberg).* Here the lipiodol X-ray

* *Cf.* Elsberg: "The False Localizing Sign of Spinal Cord Tumours," *Archives of Neurology and Psychiatry*, January, 1921, vol. v., No. 1, p. 67. See also Elsberg: "Sensory Disturbances in Tumours of Spinal Cord and Membranes," *Archives of Neurology and Psychiatry*, May, 1923, vol ix., No. 5, p. 656.

examination (*cf.* p. 149) may guard against diagnostic errors.

Addendum.—The above examination only refers to the objective disturbances of sensation. The *subjective* manifestations (pains and paræsthetic sensations) the patient will have related when giving his *historia morbi*. It must be ascertained exactly where the pains are localized, if they are constant or paroxysmal, if they have a fixed *cyclus*. Nowhere in the whole neurological examination does the *individuality* of the patient show itself as in his description of pains.

In thalamic lesions paroxysmal pain in the contralateral side is a prominent feature. In hemiplegia depending on capsula interna lesion pains are also frequent, but are chiefly localized to the joints, which have been more or less immobilized—the shoulder-joint first and foremost. In thalamic lesion the pain is chiefly localized in the trunk; there is also, as a rule, hemi-hypoæsthesia, chiefly affecting the deep sensation and accompanied by marked over-reaction to painful stimuli, and still more to cold.

This hypersensitiveness to cold is not seldom the only phenomenon of over-reaction in thalamic lesions; it is, however, also sometimes seen in tabes.

5. REFLEXES.

Testing the reflexes is one of the most important parts of the clinical examination—probably *the* most important. Its great advantage is its *objectivity*. It is not so dependent on the attention and intelligence of the patient as the motor and sensory examination. It can be carried out in unconscious patients and in children, and an attempt at simulating reflex changes is a rare occurrence and as a rule very easy to discover.

For purposes of clinical investigation we distinguish between—

I. Tendon and periosteal reflexes.
II. Cutaneous reflexes.
III. Reflexes of spinal automatism.
IV. Postural reflexes.
V. Organic reflexes.

I. **Tendon and Periosteal Reflexes (Deep Reflexes).**

(Eliciting stimulus: brisk tap on a tendon or a bony prominence.)

In order to elicit these it is necessary for the patient to relax his limbs, as voluntary fixation may prevent the reflexes from being elicited. A suitable reflex hammer is of great help in eliciting the reflexes. An ordinary wooden stethoscope with a rubber ring round the plate forms a very useful reflex hammer. The fingers may also be used, as in percussion.

When a tendon reflex is elicited normally the antagonists will also be felt to contract. In paralysis agitans and in hysteria this contraction of the antagonists may be very marked, whilst in cerebellar lesions it is generally absent.

The reflexes commonly tested are the following:*

1. *Jaw-Jerk.*—The patient opens his mouth, and his chin is grasped with left thumb and forefinger (the

* In cases of increased reflex irritability a great many more deep reflexes are found; in many such cases it is impossible to tap any bony prominence or any tendon without eliciting some jerk.

thumb on the top). A brisk tap on the examiner's own thumb results in contraction of both masseter muscles.

Reflex centre=pons.

In the upper limbs we have—

2. *Radialis Periosteal Reflex* (*Supinator Jerk*).—The arm bent at an open angle, the hand resting, semi-pronated upon the bed, the table, or the patient's knee. A brisk tap on the distal end of the radius elicits a contraction of the supinator longus (musculus brachio-radialis), and sometimes also of the biceps, with resulting flexion at the elbow-joint.

Reflex centre=sixth cervical segment.

Often, also, a contraction of the fingers is observed (C_8). Due to the different segmental representation of these different muscles a dissociated response can take place in circumscribed medullary lesions (Babinski)—*cf.* Motor Segmentation (p. 68). When this reflex is tested in a position of maximal supination, as a rule no response (except perhaps flexion of the fingers) is obtained, unless the reflex irritability is increased—*e.g.*, in pyramidal lesions; then a reflex contraction of the supinator longus and the biceps or of the triceps may be observed.

Sometimes the reflex is somewhat difficult to elicit. In these cases a brisk tap may be tried on the *volar* aspect of the inferior end of the radius, so as to cause a slight supination; this elicits a reflex pronation rebound—the *radial pronator reflex*. A similar pronation reflex can be elicited by tapping the styloid process of the ulna—*the ulnar pronator reflex*. Both these reflexes are examined in the semipronated position. When the

latter is examined in a position of maximal supination a movement of abduction at the shoulder-joint is claimed to be indicative of a central motor (? pyramidal) lesion (Marie, Bouttier, and Bailey—"supino-reflexes").*

3. *Biceps Reflex.*—Arm in the same position as for (2), but with full supination of hand. Grasp the patient's elbow with the left hand, placing the thumb on the biceps tendon. A tap on the examiner's thumb elicits contraction of the biceps.

Reflex centre=fifth and sixth cervical segments.

4. *Triceps Jerk.*—Lift the patient's arm from the bed, grasping it loosely round the overarm—slight flexion at elbow-joint. A tap on the triceps tendon just above the olecranon† elicits a contraction of the triceps, with extension of the arm.

Reflex centre=about eighth cervical segment.

The above-mentioned deep reflexes in the upper limbs (2, 3, and 4) are sometimes difficult to elicit, especially in youthful individuals of rather stout build. When, therefore, in a young muscular individual one or all of

* In the author's opinion this abduction is chiefly a "pronation in disguise." Any contraction of the pronators will, when the wrist is firmly fixed in maximal supination and the arm bent at the elbow, result in a movement of abduction at the shoulder.

The systematic search for these "supino-reflexes" is valuable, as it allows of a more objective estimation of the strength of the deep reflexes of the upper limb; but their presence is *no proof* of pyramidal lesion.

† A tap on the olecranon itself (olecranon reflex) may elicit a *flexion* at the elbow-joint (biceps contraction). This olecranon reflex (which is by no means found in all normal individuals) must not be confused with the inversion of the triceps reflex, which Souques has repeatedly noted in patients suffering simultaneously with tabes and hemiplegia.

these reflexes cannot be elicited on either side,* no importance need be attached to this, if no other pathological signs be present.

In the trunk we have—

5. *Periosteal Reflex of the Costal Margin.*—A tap on the costal margin in the nipple line elicits a local contraction of the abdominal muscles, with deviation of the umbilicus towards the costal margin, where the tap was given.†

Reflex centre = eighth and ninth dorsal segments.

In the lower limbs we have—

6. *The Knee-Jerk.*—The leg flexed at the knee-joint at an angle of 120 to 130 degrees. When the patient is sitting on a chair, he may either cross one leg over the other or, preferably, keep both feet flat on the ground. A tap on the quadriceps tendon below the patella elicits contraction of the quadriceps, frequently accompanied by a contraction of the adductor muscles on the same and on the opposite side—*homolateral and crossed adductor reflexes.* These can also be elicited by means of a tap on the condylus internus femoris.

Reflex centre = third and fourth lumbar segments.

Absence of knee-jerks constitutes Westphal's sign—one of the three "cardinal signs" of tabes.

* A difference between the two sides is always to be regarded with suspicion.

† This reflex must not be confounded with the cutaneous abdominal reflex, which belongs to a quite different order of reflexes (see later, p. 102). Thus in pyramidal lesions the periosteal reflex of the costal margin may be found to be quite brisk, whilst the abdominal reflex is diminished or even abolished.

After severe bodily exertion the author has found the patellar reflexes considerably diminished and even abolished.*

7. *The Ankle-Jerk.*—The foot at right angle with the leg. Tap on the Achilles tendon elicits contraction of the gastrocnemius and soleus muscles, with resulting plantar excursion of the foot. This reflex is most easily elicited when the patient is kneeling on a chair.

Reflex centre=first and second sacral segments.

Pronounced œdema of the legs may prevent the reflex from being elicited. It is frequently diminished in sciatica, and as a rule is absent in tabes; in the latter disease it is often abolished long before the knee-jerk.

The tabetic process frequently attacks the sacral roots first, thus causing absence of ankle-jerks, hypoæsthesia along the back of lower limbs and round the anus and genitals, Abadie's sign, sphincter disturbances. The examiner should never content himself with the diagnosis of tabes, but should try to make a segmental diagnosis of the tabetic lesion.

In some cases of *sciatica* also the ankle-jerk is diminished or lost. This diminution or loss may be found already a few days after the onset, and may, on the other hand, remain long after all pain and tenderness has disappeared, in some cases for life.

Loss of ankle-jerks and increase of deep pressure pain in the calf muscles may sometimes be the only signs of *"latent" alcoholic polyneuritis.*

Ankle Clonus.—The foot is briskly pushed up in extreme dorsiflexion and held there by a moderate

* *Cf.* Monrad-Krohn: "Reflex Changes after Severe Bodily Exertion," *Norsk Magazin for Lægevidenskaben*, 1919, No. 7. (Examination of knee-jerks in forty-nine men before and after 50 kilometres ski race.)

pressure; a clonic contraction of the gastrocnemius and soleus muscles results.

This phenomenon is nothing but an exaggerated ankle-jerk, elicited in a different way. All tendon reflexes may become clonic when the reflex irritability is increased. This is most often due to a pyramidal lesion, but may also be found in "functional" conditions.

Patellar Clonus.—The leg extended, the patella is seized and pulled briskly downwards; it then starts "dancing" (clonic contractions of quadriceps).

In examining the tendon reflexes, it will sometimes be found that the patient has difficulty in relaxing the limb. His attention must then be diverted from it. A practical way of doing this is to let him make a simultaneous muscular effort with his hands. The most common method is indicated by Jendrassik: the patient hooks the flexed fingers of either hand round the tips of the other, and pulls either constantly or in jerks; if in jerks, the eliciting tap is given on the tendon immediately after the patient starts pulling.*

In pyramidal lesions the tendon and periosteal reflexes are as a rule exaggerated below the lesion. In complete division of the cord, however, the deep reflexes are as a rule abolished, at least for a time. When the lower limbs are the seat of a strong flexion contracture, it is often impossible to elicit the deep reflexes of the lower limbs. Whether this is due to the posture only is debatable. Déjerine claims that in cord lesions, where there is complete sensory loss—both deep and superficial—the

* For the clinical note-taking it is convenient to record a feeble reflex with +, a reflex of average strength with ++, a reflex of more than average strength with +++, and a clonic reflex with ++++; *e.g.*, biceps reflex: $r.+++ > l.++$.

deep reflexes are invariably lost. The author has seen striking exceptions to this rule, which cannot be regarded as absolute.

In cerebellar lesions the deep reflexes are often *pendular*, due to diminution of tonus and loss of accompanying reflex contraction of the antagonists; this is most noticeable in the triceps and knee jerks.

In addition to the tendon and periosteal reflexes the different muscles and nerves are tapped lightly to test their mechanical irritability.

In *reflex nervous disorders*, according to Babinski and Froment, the mechanical irritability of the muscles is, as a rule, considerably increased. This increase is most marked locally at the site of the reflex-contracture, -atrophy, or -paresis, and shows a particular predilection for the distal parts of the limbs (thenar and hypothenar eminences).

Also, in *tetany* there is an increased mechanical irritability both of nerves and muscles. The Chvostek phenomenon is commonly described as a unilateral facial contraction elicited by tapping the facial nerve; as a rule it is most easily elicited by a gentle tap on the cheek just below the malar bone.

It is practically always present in tetany in children, but can hardly be considered pathognomonic of tetany, as it is frequently found in children *not* suffering from tetany. It is a question if it is not a reflex (tendon or periosteal reflex).

The pectoralis major frequently shows a distinct mechanical irritability; no distinct diagnostic importance attaches to this phenomenon.

Another phenomenon characteristic of tetany is the *Trousseau phenomenon:* pressure (digital or by means of an elastic bandage round the arm) in the region of the internal bicipital sulcus produces, in individuals suffering with tetany, a typical tetanic contracture (tonic spasm) of the hand. The contracture appears after a period of latency varying from thirty seconds to four minutes. If after five minutes' pressure it has not appeared, the test is considered to be negative.

By means of *hyperventilation* manifestations of tetany can experimentally be elicited in most normal individuals. In many epileptics these manifestations are followed (or interrupted, or even preceded) by epileptic manifestations (according to *Foerster* in about 50 per cent. of all epileptics).*

For the sake of hyperventilation the patient is placed in the recumbent position and instructed to breathe as deeply and quickly as he can. After a period varying up to fifteen or twenty minutes the above phenomena (of tetany or epilepsy) occur.

II. **Cutaneous Reflexes (Superficial Reflexes).**

(Eliciting stimulus: Stroking the skin by means of a pin or a pencil.†)

The most important of the cutaneous reflexes is—

1. *The Plantar Reflex.*—On stroking the sole with a pencil or a pin, *normally* a *plantar* movement of the big toe ensues; in *pyramidal lesions* an *extensor*—viz., dorsal—movement ensues (sometimes accompanied by

* *Cf. Zentralblatt für die gesammte Neurologie und Psychiatrie,* 1924, Bd. 38, p. 298.

In my clinic only three of twenty-nine epileptics gave a positive hyperventilation test.

† The two essential elements are, according to Strümpell, 'Zeitliche und örtliche Summation."

a spreading movement of the other toes—"signe de l'éventail"). This extensor response is, as a rule, slower and lasts longer than the normal plantar response; in peripheral paralysis, as in peripheral anæsthesia of the sole, no response is obtained (*e.g.*, in many cases of poliomyelitis and tabes).

The sole is stroked in a forward direction, and both along the lateral and the median border. The pathological extensor response is most easily elicited from the lateral part of the sole, the normal from the median part. When extensor response is only elicited from the lateral border, it is still a sign of pyramidal involvement, as normally even from this part of the sole a plantar movement should be elicited ("formes frustes"—Babinski).*

Babinski has shown that the dorsal type of plantar reflex (plantar reflex with extensor response) is a reliable sign of pyramidal lesion. The normal plantar reflex with flexor response has probably a cerebral reflex arc, the efferent part of which is the pyramidal tract. When this is affected, a purely spinal reflex mechanism brings out the extensor response, which is the original, and in the first year of life the normal, response.

In the first eight weeks 92 per cent. give extensor response; in the first year 77 per cent.; and in the second and third year 5 per cent.†

In young children the plantar reflex therefore has a limited diagnostic value.

* A doubtful extensor response may be rendered definite by a hypodermic scopolamin injection (0·25 to 0·5 milligramme).

† According to Bersot and Lantuéjoul, the plantar reflex is at birth flexor, but very soon becomes extensor.

It is convenient to record the normal plantar reflex with an arrow pointing downwards, thus:

Plantar refl. ↓

and the extensor plantar response with an arrow pointing upwards, thus:

Plantar refl. ↑

Oppenheim and Gordon have each described a substitute for Babinski's plantar reflex. The first consists in a dorsal movement of the big toe in response to a firm stroke along the median border of the tibia; the latter in a dorsal movement of the big toe elicited by pinching the calf muscles. Both are significant of a pyramidal lesion. Both belong (as does the inverted plantar reflex) to the order of reflexes of spinal automatism (see later).

When in peripheral paralysis (*e.g.*, poliomyelitis) the *short* flexor muscles of the toes (flexor hallucis brevis, abductor hallucis, and adductor hallucis) are paralyzed, whilst the extensors are intact, the plantar reflex gives an extensor response. This extensor response is, of course, no sign of pyramidal lesion.* For the valuation of this reflex it is therefore necessary to consider the voluntary motor power of the big toe, particularly of its proximal phalanx, where the above-mentioned muscles are inserted.†

* With my assistant, Dr. Lossius, I have described two cases of this kind (*cf. Norsk Magazin for Lægevidenskaben*, 1921, 11).

† With the exception just mentioned, the dorsal response of the plantar reflex may be said to be a reliable indication of pyramidal lesion. On the other hand, it cannot be claimed that a normal response of the plantar reflex necessarily proves that there is no pyramidal involvement. Thus in amyotrophic lateral sclerosis the plantar reflexes sometimes remain normal. *Cf.* Monrad-Krohn: "Les réflexes plantaires dans la sclérose latérale amyotrophique," *Revue neurologique*, 1925, i., No. 6, p. 831.

In *complete* transverse lesion of the spinal cord the plantar reflex, according to Guillain, always gives a *plantar response,* which, however, differs from the normal plantar reflex by its *slowness.*

2. *The Abdominal Reflexes.*—Stroking the abdominal wall with a pin or a pencil well to the side of the middle line elicits a homolateral contraction of the abdominal muscles, resulting in deviation of the linea alba and the umbilicus. The reflex may be elicited both in a standing and in a recumbent position. It is best to have the patient lying flat on his back with his head resting on the pillow; he is asked to relax his abdominal muscles as much as possible—if necessary, to take a deep breath. The end of the inspiration, when the respiratory muscles are beginning to relax, is the phase in respiration when the abdominal reflexes are most easily elicited. As a rule, however, there is not much difference between the different respiratory phases in this respect.

The maximum contraction is normally elicited at the level of the stimulus, and we distinguish between—

(*a*) An epigastric reflex elicited by a stroke above and along the costal margin (*cf.* Fig. 28).

This results in a retraction of the epigastrium, the lateral deviation of the linea alba being less marked in this region. In individuals with fat and flabby abdominal walls the epigastric reflex is often the only one present of all the abdominal reflexes.

(*b*) A supra-umbilical abdominal reflex elicited by a horizontal stroke towards the middle line above the umbilicus.

(*c*) An umbilical abdominal reflex elicited by a horizontal stroke at the level of the umbilicus; and

(*d*) An infra-umbilical abdominal reflex elicited by a horizontal stroke below the umbilicus.

When the abdominal wall is fat and flabby, the stroke often sets up an undulating movement in the panniculus adiposus, which makes it difficult to decide whether there is any muscular reaction at all. In these cases it is to be recommended to stroke with

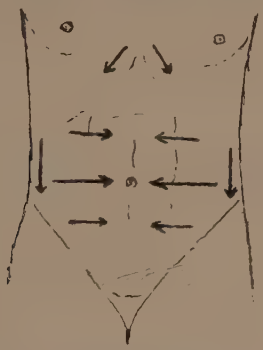

FIG. 28.

The arrows in the above diagram indicate the strokes employed for eliciting the various abdominal reflexes.

the pin vertically between the nipple line and the anterior axillary line (*cf.* Fig. 28); a deviation of the umbilicus or the linea alba can then be relied upon to be the result of muscular contraction—can, in other words, be regarded as an abdominal reflex. It will be found practical to draw a line with a skin-pencil from the xiphoid process to the symphysis. This makes the deviation of the middle line more noticeable.

The reflex arc of the abdominal reflex reaches the cerebrum (probably even the cerebral cortex).

The pyramidal tract forms the efferent part of the reflex

arc; the afferent part probably consists of uncrossed centripetal fibres in the posterior columns of the cord (the spinothalamic tracts),* or may even consist of a double path of conduction.

When the abdominal reflex is exaggerated, the reflexogenous zone is enlarged; in some cases an abdominal reflex can thus be elicited from the upper third of the thigh. Exaggeration of the abdominal reflexes is frequently found in functional disorders, where often "the umbilicus chases the pin." The abdominal reflex is a cerebral reflex. It is diminished or absent in pyramidal lesions as well as in peripheral motor lesions. Sensory loss—whether peripheral or central—must be profound before the abdominal reflex is affected. In most cases of tabes the abdominal reflexes are very brisk; only when abdominal hypoæsthesia approaches complete anæsthesia the abdominal reflex disappears. In disseminated sclerosis the abdominal reflexes often disappear at an early stage;† in hysteria unequal abdominal reflexes with corresponding sensory change over the abdomen is quite a frequent stigma; in paralysis agitans and chorea the abdominal reflexes are, as a rule, peculiarly brisk—with hemiplegic distribution brisker on the affected side; in partial paralysis of the abdominal wall (as frequently seen in poliomyelitis) a stimulus in a paralytic area elicits a *heterosegmental* abdominal reflex—*i.e.*, the maximum contraction does

* *Cf.* Monrad-Krohn and Sæthre: "Clinical Observations bearing upon the Centripetal Path of the Abdominal Reflex," *Norsk Magazin for Lægevidenskaben*, 1921, No. 2, p. 135.

† *Cf.* Monrad-Krohn: "Les réflexes abdominaux dans la sclérose en plaques," *Revue neurologique*, 1924, i., No. 6, p. 707.

not correspond to the level of stimulus, as it necessarily must take place outside the paralytic area.

It has to be kept in mind that the abdominal reflex may also be affected by acute abdominal conditions.*

It has further to be kept in mind that by no means every reflex contraction of the abdominal wall, elicited from the abdominal region, is to be regarded as identical with the normal abdominal reflex, which has a cerebral reflex arc (of which the pyramidal tract forms the centrifugal part). One must distinguish between the normal cutaneous abdominal reflex, which is chiefly unilateral with more or less marked deviation of the umbilicus and the linea alba, and ordinarily elicitable only by stroking, and pathologic abdominal reflexes.

These pathologic abdominal reflexes belong to the order of "reflexes of spinal automatism," and are generally associated with other such reflexes (flexion or extension reflexes of the lower limbs). When, however, the latter are not pronounced, the distinction between the normal and abnormal abdominal reflexes may be difficult, and one must rely on the following points:

The pathologic abdominal reflex has a greater tendency than the normal one to bilateral contraction. It is, therefore, more a "retraction reflex," while the normal one is more a "unilateral deviation reflex."

The pathologic abdominal reflex has sometimes a distinctly prolonged period of latency.

It is less elective as regards the stimulus required.

The practical importance of this distinction is chiefly in connection with segmental focal diagnosis. When a pathologic abdominal reflex is wrongly deemed to be a normal one (and in the absence of sensory disturbances the diagnosis

* For a more detailed description of the abdominal reflexes and their clinical value the reader is referred to the author's monograph, "Om Abdominal-Reflexerne," Christiania, 1918, of which a summary in English is given in *The Practical Medicine Series*, vol. viii., edited by Professor Bassoe, Chicago, 1919.

of the level of the lesion depends mainly on motor loss, often slight, and reflex findings), such a mistake may lead to the faulty assumption of a low dorsal or lumbar lesion when the lesion really is situated at a much higher level.

While the existence of a normal abdominal reflex depends on the integrity of the corresponding pyramidal fibres (central efferent reflex path), the pathologic abdominal reflexes just described are independent of the pyramidal tract (*cf.* also p. 110).

3. *The Cremasteric Reflex.*—A stroke along the inner aspect of the thigh elicits a brisk contraction of the homolateral cremasteric muscle.

Often this reflex is most easily elicited from the *posterior* parts of the inside of the thigh.

The superficial reflexes all have cerebral reflex arcs;* we therefore cannot speak of spinal reflex centres for these reflexes; but it is well to know that the peripheral parts of the reflex arcs enter and leave the spinal cord—

for the abdominal reflexes at the seventh to twelfth dorsal segments;
for the cremasteric reflex at the first lumbar segment;
for the plantar reflex at the first and second sacral segments.

Closely related to the cutaneous reflexes are the *corneal,* the *conjunctival,* and the *pharyngeal* reflexes. In the two former, closure of the eyes is elicited by touching the

* There may, however, be certain differences as to the exact anatomy of these central reflex arcs. Clinical experience, at any rate, does not show that complete parallelism in the behaviour of the abdominal and cremasteric reflexes which one would expect, were the reflex arcs quite similar. *Cf.* Monrad-Krohn and Kornfeldt: "The Cremasteric Reflex in its Relation to the Abdominal Reflex," *Archives of Neurology and Psychiatry,* April, 1925, vol. xiii., No. 4.

cornea or the conjunctiva. For this purpose a piece of soft blotting-paper or a wisp of cotton-wool is conveniently used. The conjunctival reflex is often absent in normal individuals; but the corneal reflex is a constant phenomenon, the absence of which has pathological significance. In the pharyngeal reflex, touching of the mucous lining of the pharynx elicits a complex movement of retching.

This latter reflex may be absent in hysteria.

III. Reflexes of Spinal Automatism ("Réflexes de Défense").*

These are reflexes which are in the normal condition subjected to such inhibition that, with the exception of the flexion reflex, as a rule they cannot be elicited. In central motor lesions—and particularly in spinal lesions—they may be elicited with such ease and with such strength that they must be considered to be of great diagnostic value. The reflexes may, perhaps, in some respects be regarded as clinical homologues of reflexes seen in decerebrate and spinal animals (*cf.* Sherrington, Pierre Marie and Foix, Babinski, Walshe, Riddoch, and others).

* These reflexes, some of them already observed and described by Marshall Hall, have been given a number of different names by the different authors—viz., "réflexes à court trajet exclusivement médullaire," "irritation reflexes," "atypical cutaneous reflexes," "wild reflexes," "réflexes cutanés inférieures," and many others. A really good name for them has not been found yet. *Cf.* Monrad-Krohn: "Reflexundersögelser," *Norsk Magazin for Lægevidenskaben*, 1924, No. 1, p. 31, and Axel Oewre: "Spinal-automatiske reflexer," Oslo, 1924. For a completer survey of these reflexes the reader is referred to Babinski's lecture, "Réflexes de défense," *Revue neurologique*, 1922, No. 8.

1. *Flexion Reflex of the Lower Limbs* (*Phénomène des Raccourcisseurs*).—A nocuous stimulus applied to the distal part of the lower limbs elicits a complex reflex movement consisting of flexion at the hip-joint, flexion at the knee-joint, and dorsiflexion at the ankle-joint ("triple flexion"). *Normally the dorsiflexion at the ankle-joint occurs when the reflex is elicited from the sole of the foot only.* When a stimulus applied anywhere else elicits dorsiflexion at the ankle-joint, it is pathological and indicative of a pyramidal lesion. In normal individuals, if at all present, the movement is a quick one, and as a rule accompanied by flexion of the toes. In central motor (pyramidal) lesions the movement is slower and, as a rule, accompanied by a dorsiflexion of the toes. The flexion is elicited by all sorts of nocuous stimuli: pinprick, pinching of the skin,* thermic stimuli,† strong, deep pressure, excessive passive flexion of toes (Marie and Foix).

In order to decide whether the flexion reflex is pathologically exaggerated or still within the limits of normal variation, the best way of proceeding is the following: Pinch (or prick) the skin of the *dorsal* aspect of the foot vigorously. If *dorsal* movement of the foot ensues, the flexion reflex may safely be regarded as pathologically exaggerated. When the pyramidal tracts are intact, this form of reaction never occurs (Babinski).

In spinal lesions two types occur—

(*a*) Uniphasic motor reaction—viz., flexion only (this is the only response possible in complete division of the cord).

(*b*) Biphasic reaction—viz., flexion followed by extension (only possible in incomplete division of the cord).

* Babinski recommends pinching the skin of the anterior aspect of the leg just above the ankle. This has the advantage of affording an opportunity of observing the toe movements, which cannot be observed when Pierre Marie's method of eliciting the reflex by passive flexion of the toes is employed. But generally it is found that this latter method more readily elicits the reflex.

† The sudden uncovering of the legs often elicits a brisk flexion reflex of both lower limbs, which, when occurring in a completely paraplegic patient, often startles the inexperienced.

The upper border of the reflexogenous zone in a number of cases corresponds to the lower limit of the spinal lesion, and may thus be of importance for the focal diagnosis. It does not, however, always reach as high up, but the reflexogenous zone never extends upwards beyond a line corresponding to the lower limit of the lesion.

The flexion reflex is the most important of the reflexes of spinal automatism. It is most pronounced in those cases where, as a result of a spinal lesion, a flexor contracture has developed. This contracture may, according to Babinski, be regarded as a fixed flexion reflex (un phénomène des raccourcisseurs fixé—Foix).

Just as a brisk flexion reflex is a prominent feature of the usual flexor contracture (the tendon reflexes being frequently diminished or absent), so increased deep reflexes are an equally characteristic feature of extensor contracture, which, being the usual form for contracture in hemiplegia,* is probably a sign of *pure* pyramidal involvement. The latter form is therefore indicative of a less severe lesion than is the flexor contracture, which is probably due to extrapyramidal as well as pyramidal involvement. Babinski defines this extensor contracture as "tendino-reflexe," and the flexion contracture as "cutaneo-reflexe" (*cf.* van Gehuchten's term for reflexes of spinal automatism—viz., réflexes cutanés inférieurs).

2. *Crossed Extension Reflex.*—Stimulation of the sole of one foot causes (besides flexion of the homolateral leg) extension of the contralateral leg (indicative of *in*complete spinal lesion). Patient is lying flat on his back with both legs flexed.

3. *Extensor Thrust.*—Active extension of the limb when the distal portion of the sole of the foot is pressed upwards, the limb first having been passively flexed. Extension may be followed by flexion, and a *stepping*

* It has to be remembered, however, that cerebral lesions (probably by involvement of the central ganglia) may sometimes produce flexor contracture (*cf.* Alajouanine: *Sur un type de paraplegie en flexion d'origine cérébral*, Paris, 1923).

movement in the two limbs may thus be started in some cases. (This never found in complete division of the cord—Riddoch.)

4. *Abdominal Contraction.*—If the lesion be above D_{12}, it may be found that a more or less extensive portion of the abdominal wall takes part in the flexion reflex. As the reflexogenous zone may often extend over part or the whole of the abdominal wall, a stimulus applied to the abdominal area will in these cases sometimes elicit an abdominal contraction; this contraction is of a type different to the normal abdominal reflex, as it is slower and has a greater tendency to become bilateral (a general contraction of the abdomen).* It may be accompanied by a homolateral flexor (or extensor) reflex of the lower limb, even when elicited from the abdominal region (*cf.* also p. 105).

It should be carefully noted how high up this abdominal contraction extends. The upper limit of the reflex contraction indicates the lower limit of the spinal cord lesion. When this latter is situated in the lower dorsal cord, the upper limit of the contraction in lean individuals is often fairly distinctly defined in the rectus abdominis muscle.

5. A complex response to nocuous stimuli applied to the lower limbs, consisting of (*a*) flexion reflex, as described above, (*b*) evacuation of the bladder, and (*c*) sweating from the cutaneous segments below the lesion, has been described by Riddoch as *mass reflex*—a reflex response typical of spinal lesions, when the period of shock has passed off and before septicæmia, so frequently arising from complications (bedsore, urinary infection), has set in and destroyed the reflex irritability of the spinal cord. This mass reflex is not only of diagnostic value, but may also be made use of therapeutically in re-educating the bladder function. The reflexogenous area of the mass reflex is not confined to the

* The distinction may, however, present difficulties. *Cf.* Monrad-Krohn: "On Reflexes of Different Order, Elicitable from the Abdominal Region," *Archives of Neurology and Psychiatry*, June, 1925, vol. xiii., No. 6, p. 750.

surface of the body, but comprises also the bladder, by distension of which the whole complex reflex may be elicited. This reflex is always an indication of grave spinal injury.

Also for the upper limbs similar reflexes have been described (Böhme, 1917, Riddoch and Farquhar Buzzard, 1921), viz.: a *flexion reflex*—centre of reflexogenous zone in the palm of the hand—and an *extension reflex*—centre of reflexogenous zone in the axilla and adjacent region of upper arm.

In the ordinary routine examination it is unnecessary to test for all these reflexes; but always the skin of the dorsal aspect of the foot or the ankle should be pinched or pricked to see if this elicits a dorsiflexion at the ankle-joint (*pathological shortening reflex*). If this be the case, one may conclude that there is a central motor lesion of pyramidal origin.

A simple exaggeration of the normal shortening reflex (flexor reflex) of the lower limb is found in many neurotic patients and in diffuse cortical lesions, as, *e.g.*, dementia paralytica.

IV. **Postural Reflexes.**

" Standing " and " Righting " Reflexes.—These reflexes (chiefly studied by Dutch investigators, Magnus, de Klein, and others) are complex reflexes, aiming at maintaining or re-establishing the standing position.

Experimental work has discovered five groups of such reflexes: (1) optic righting reflexes; (2) labyrinthine righting reflexes, acting upon the head; (3) righting reflexes, arising in the deep tissues of the body and acting upon the head; (4) righting reflexes, arising in the deep structures of the neck and acting upon the trunk and limbs; (5) righting reflexes, arising in the deep tissues of the body wall and acting upon the limbs and trunk.

The posture of the head (" the leading segment," Sherrington) has the greatest influence on the attitude of the rest of the body and " in a decerebrate animal we can vary the body posture at will, using the head as a handle " (Walshe).

Of the postural reflexes just mentioned, it is (4) the

neck reflexes, which are the most important in the clinic: rotation of the head causes an extensor movement or an increase of extensor tonus in the arm on the side to which the face is turned, and a flexor movement or increase of flexor tonus in the opposite arm. The extension is, as a rule, combined with supination, the flexion with pronation. These movements are only seen in limbs "released from pyramidal control"—*i.e.*, in arms affected by paresis of pyramidal origin (they are particularly pronounced in Little's disease). The postural reflexes are missing in extra-pyramidal lesions of the paralysis agitans type. (It is readily seen that these postural reflexes are something quite different, partly even contrary in their actions, to the "postural reflexes of Foix"; *cf.* footnote on p. 56.)

V. **Organic Reflexes.**

In taking the patient's history it will have been found out whether there be *automatism of the bladder*—viz., the bladder voids itself at intervals involuntarily, with or without the knowledge of the patient. When the voluntary control is deficient we have *precipitate micturition*—a state half-way between automatism of the bladder and the normal state (this frequently seen in disseminated sclerosis); the patient is aware of the beginning bladder reflex, but has difficulty in arresting it.

If there is *dribbling incontinence* a distended bladder will, as a rule, be found; the detrusor reflex is here lost, and the sphincter resistance is overcome by the mere mechanical factor of overfilling (*ischuria paradoxa*).

It is well to decide the amount of residual urine by passing a catheter after the patient has made an effort to empty his bladder; at the same time, by filling the bladder, it can be determined if there be any detrusor reflex and at what degree of filling it occurs.

Disturbances in the function of the rectum will also have been elicited in taking the *historia morbi*.

The *internal anal reflex* is tested by inserting a finger in the anus. When this reflex is present, the finger is tightly grasped by the sphincter. In case of loss the

anus remains open—"yawns"—for several seconds after the withdrawal of the finger.

The above are the organic reflexes of most practical importance; but there are others that occasionally will have to be examined, although not forming an integral part of the *routine neurological* examination.

The *oculo-cardiac reflex* consists in a slowing down of the pulse-rate, elicited by pressure on both eyeballs backwards into the orbits.

Normally an immediate slowing of the pulse-rate amounting to 7 to 8 beats in the minute is found. As soon as the pressure is relieved, the pulse resumes its normal rhythm. This reflex is frequently found to be exaggerated in exophthalmic goitre, and abolished in many cases of tabes and under the influence of atropine.

The trigeminal nerve contains the centripetal path, and autonomic fibres of the vagus form the centrifugal part of the reflex arc.

The "vagotonic" type of individual has a pronounced oculo-cardiac reflex; in the "sympathetico-tonic" type the reflex is diminished or absent.

The *scrotal reflex* must not be confused with the cremasteric reflex described above. The scrotal reflex is a purely autonomic reflex, and its motor response much slower than that of the cremasteric.

The patient stands with his legs wide apart, the scrotum hanging free. Some cold object (*e.g.*, tuning-fork) is placed in the perineum, and after a short interval of a few seconds slow, worm-like contractions of the dartos will be noticed. The reflex can also be elicited by stroking the perineum repeatedly (six to seven times) with a pin.

The *cutaneo-gastric reflex* is elicited by stroking the skin gently along the left costal margin; the motor reaction consists in a gastric contraction, which can be observed by auscultation after about a minute's stroking.

The *pilomotor reflex* consists in a contraction of the erectores pilorum ("goose-flesh"). It may be elicited as a local, "idiomotor" phenomenon, or through the intermediary of the central nervous system (sensory nerve, posterior roots, ascending or descending spinal fibres to

pilomotor "centres" in dorsal cord (D_1 to L_2)—*cf.* Fig. 54—from these centres efferent paths through the sympathetic nervous system), and then extends to wider parts of the surface of the body. The eliciting stimulus can be gentle stroking, tickling, or cold. The regions from which the pilomotor reflexes are most readily elicited are the nape of the neck and the subaxillary regions. In many patients pilomotor reflexes are easily elicited by pinching the superior border of the trapezius muscle. Pilomotor reflexes are as a rule also elicited in connection with the reflexes of spinal automatism.

Numerous sources of errors have to be guarded against. First of all against the influence of cold air when the patient is examined in a cold room. The pilomotor reflexes should, therefore, always be investigated in a well-heated room. It should also be kept in mind that emotional stimuli (fright or anger) may cause a general pilomotor reaction. Also unpleasant aural sensations (grating noises) may produce pilomotor reactions in many individuals.

The area of the reaction ("goose-flesh") and the stimulus (its kind and localization) must both be accurately recorded.

When the eliciting stimulus is applied to one side of the body only, the pilomotor reflex response is unilateral (*i.e.*, homolateral), and does not go beyond the middle line. The reflex is a slow one; it appears after a varying period of latency, difficult to measure, as the eliciting irritation must, as a rule, be continued for some time. The pilo-contraction itself is completed in seven to ten seconds, and after the contraction being maintained for some time, greatly varying in different individuals, the pilomotor muscles relax again (this relaxation takes, according to André Thomas, about double the time of that of the contraction—viz., fourteen to twenty seconds). The reaction does not appear uniformly and simultaneously all over the body. It is most pronounced on the trunk, where, when elicited from the neck or the axillary region, it appears as a descending reaction (the goose-flesh extends from above downwards), and on the extensor sides of the extremi-

ties, the distal parts of which show little reaction. According to the seat of any lesion, blocking the reflex arc, the pilomotor reflexes will be missing in the corresponding regions (*cf.* Fig. 54, p. 192). Symmetrical parts of the two halves of the body should always be carefully compared as to the pilomotor reflexes.

When the pilomotor reflexes are elicited by means of a bilateral stimulus (*e.g.*, by tickling the nape of the neck), the pilomotor reaction should be completely identical in the two halves of the body.

In many individuals the pilomotor reflexes are very easily exhausted.

As a diagnostic sign of reflex nature "*Forced Grasping and Groping*" may conveniently be mentioned here (W. J. Adie and Macdonald Critchley).* It is supposed to be a sign of a lesion in the superior posterior part of the contralateral frontal lobe.

The grasping reflex is elicited by touching the palm, preferably one of the metacarpo-phalangeal joints; the hand clutches the observer's fingers tightly, and the grip may even resemble the myotonic grip (*cf.* p. 60).

When a finger-tip or the palmar surface of the hand or of the fingers is touched, the hand assumes a grasping attitude and moves towards the stimulus. The patient should keep his eyes shut.

Similar reactions may be observed in very young normal nfants (? before the development of frontal inhibition).

Reflex Formulæ.

1. *Pyramidal Lesion*—

   (*a*) Extensor plantar reflex. ↑

   (*b*) Diminished or absent abdominal reflexes.

   (*c*) Increased tendon and periosteal reflexes.

* Also described by Schuster and Pinéas ("*Zwangsgreifen*," "*Nachgreifen*"), Kleist ("*Greifreflex*"), and Janichewski ("*Réflexe de préhension*").

(*d*) Pathological shortening reflex—*i.e.*, dorsiflexion at ankle-joint elicited by pinching dorsal aspect of foot or ankle.

2. *Peripheral Lesion.*—Diminution or loss of all reflexes.
3. *Hysterical Lesion*—
   (*a*) Plantar reflex normal. ↓
   (*b*) As a rule, brisk abdominal reflexes ("umbilicus chases the pin"); often difference between the two sides, with corresponding sensory difference.
   (*c*) Brisk tendon and periosteal reflexes.
   (*d*) No pathological shortening reflex.

4. In a certain number of cases of *extrapyramidal* motor lesions (paralysis agitans, chorea) the following reflex formula is found—
   (*a*) Plantar reflex normal. ↓
   (*b*) Increased abdominal reflexes.
   (*c*) Retarded or diminished tendon reflexes.
   (*d*) No pathological shortening reflex.

6. TROPHIC AND VASOMOTOR STATE—CIRCULATION.

After the reflex examination a general survey of the patient should follow, particularly with a view to discovering any *trophic disturbances* present (bedsores, arthropathies, spontaneous fractures). The examination of the internal organs in the usual way is conveniently carried out here.

The *blood-pressure*—determined by the Riva Rocci method—is of great practical importance, particularly in connection with apoplexy, manifest or imminent. It should be noted if the examination is followed by the appearance of multiple petechiæ in the skin distal to

the cuff, an indication of weakness of the vessel walls or of certain forms of hæmorrhagic diathesis (thrombopenia).*

The *vasomotor system* must be carefully observed. Pronounced vasomotor disturbances, cyanosis, and hypothermia often accompany central and particularly peripheral motor paralyses.

Local cyanosis and hypothermia in cold weather are prominent features in the symptomatology of "reflex nervous disorders" as defined by Babinski and Froment. In dementia præcox cyanosis and hypothermia are commonly found in the distal parts of all four limbs.

The *arterio-capillary tension* can easily be determined by means of Gärtner's tonometer. A hollow rubber ring connected with a manometer is applied to a finger (or a toe). The part of the finger distal to the rubber ring is rendered bloodless by means of a rubber band tied round it. The ring is then blown up *ad maximum*, the rubber band is taken off, and the pressure in the rubber ring gradually reduced till the blood returns to the distal part of the finger, which then becomes pink again. When this pink colour returns the pressure is read off on the manometer—this gives the arterio-capillary tension.

The *vasomotor reaction* to pin scratches must also be observed; dermographism must be noted.

* For description of a typical case of thrombopenia with spinal lesion, observed in my clinic, see K. Evang: "A Case of Essential Thrombopenia with Hæmatomyelia," *Acta Psychiatrica et Neurologica*, 1928, vol. iii., fasc. i.

7. THE STANDING POSITION.

At the end of the clinical examination it is noticed whether the patient can *stand* and *walk*. Can he do this by himself, or has he to be supported on one or even both sides?

If he cannot stand unsupported, does he always tend to fall to the same side? If he can, does he assume any peculiar attitude? A paralysis agitans patient thus shows a characteristic attitude: stooping, with head and trunk bent forward, arms flexed and pronated, knees slightly bent (*cf.* Fig. 29). A similar attitude is frequently seen in involutional melancholia. In mania, on the contrary, the patient usually holds himself very erect. In myopathy with affection of the erector trunci a marked lordosis generally develops. Dementia præcox presents a rich variety of weird postures.

If he can stand unsupported *Romberg's sign* can be tested for, asking the patient to put his feet close together and close his eyes. If the patient sways, Romberg's sign is said to be present. It should be noted whether he just starts swaying without having to move his feet, if he manages to keep his balance by simply spreading his feet a little, or if he has to be supported in order to avoid a fall, also if he always falls in the same direction. Care should be taken to let the patient be in perfect balance before he is asked to shut his eyes. Romberg's sign is one of the three "cardinal signs" in tabes, as already mentioned, but as all of these signs, it may occur also in other conditions ("signum unum, signum nullum").

In neurotic patients (particularly women) the ordinary Romberg test is sometimes found positive, whilst it promptly becomes negative when the patient's attention is engaged by some other simultaneous test. The author generally lets the patients carry out the finger-nose test in the Romberg position. The patient's attention is then, as a rule, solely absorbed in the finger movement. In a tabetic patient this complication of the test will, on the other hand, make the patient betray his unsteadiness all the more.

The patient may also be asked to stand on one leg with his eyes shut (Fournier's test); but far from all normal individuals can do this. The patient should first stand on the one, then on the other leg, and any difference should be noted.

The patient's posture in the recumbent position is also noticed. When in bed, does he always lie on the same side (as frequently seen in vestibular and cerebellar lesion) ?

8. THE GAIT.

The student has to be seriously warned against the dangerous habit of making "lightning diagnoses" from the patient's gait. The gait should be closely observed and analyzed, and this, in *conjunction with the other findings*, is often most helpful in the diagnosis.

When studying the gait it is a great advantage to have the patient stripped or in a tight bathing suit and to let him walk in a *large* room.

The observer should direct his attention not only to the movements of the legs, but also to the arm movements normally accompanying the gait Both in pyramidal and extrapyramidal motor lesions these arm

movements often disappear. It is also very important to observe the patient's eyes. Does he look down on the ground? Some ataxic patients keep their eyes anxiously fixed on the ground all the time when they are walking (*cf.* Fig. 30).

A patient with a foot-drop will have to compensate by means of an exaggerated flexion at the knee—*steppage.*

Hyperextension at the knee-joint may be due to either tabetic hypotonia or peripheral paresis of the muscles of the thigh (often seen in poliomyelitis).

When the glutæi are deficient (*e.g.*, dystrophia musculorum) we find a waddling gait.

A tabetic patient with ataxia of the legs will walk unsteadily, with jerky, irregular steps, his feet shooting out in all directions.

In *hemiplegia* the patient will swing his paretic leg in an arch instead of lifting the foot (*circumduction*)* (*cf.* Figs. 31 and 32), or he will drag the paretic foot on the ground.

In *cerebellar diseases* the patient will walk in a reeling way ("titubatio"), or in an asynergic way—viz., the patient moves his legs in jerky movements without being able to effect the complex tonic innervation which keeps the weight of his body over his feet—"the patient runs away from his equilibrium."

The opposite occurs in many cases of *paralysis agitans*, where there is *propulsion*—viz., the patient, once started, has difficulty in stopping—"the patient is running after his equilibrium." The same phenomenon may be observed when the patient is walking backwards—*retropulsion.*

In *cerebral diplegia* in infants we generally find scissor gait, and not infrequently an uncontrollable stepping

* It will frequently be found that the reason for this circumduction is not that the patient is unable to lift his foot at all, but that he cannot lift it *quickly enough.* (Slowness of movement is one of the chief characteristics of most pyramidal lesions, as mentioned before.) The associated movement of abduction (*cf.* Saethre's observations, mentioned on p. 70) may be another determining factor—or perhaps the chief one.

FIG. 29.— CASE OF PARALYSIS AGITANS.

Typical attitude.

FIG. 30. — TABETIC PATIENT WITH CONSIDERABLE ATAXIA.

Unsteady gait. Hyperextends at knee-joints. Keeps his eyes anxiously fixed on the ground all the time when walking.

FIG. 31.— CASE OF LEFT HEMIPLEGIA.

Circumduction of left leg. Typical attitude of left arm.

To face page 120.

FIG. 32.—CASE OF LEFT HEMIPLEGIA.
Circumduction of left leg. Typical attitude of left arm.

FIG. 33.—SAME PATIENT AS IN FIG. 32, FROM BEHIND.
Supination of left foot when he lifts it from the ground (Strümpell phenomenon, *cf.* p. 70).

To face page 121.

reflex at every attempt at walking; in adults generally *marche à petits pas.*

In *hysteria* we find all manner of disturbances of gait; they always show a marked incongruity to the other physical findings, and have a tendency to assume a certain grotesque character. The hysterical imitation of organic disturbances has to be kept in mind. The most pronounced form of hysterical motor disorder is *astasia-abasia,* when the patient can neither stand nor walk. Two forms are distinguished—viz., *paralytic astasia-abasia,* in which the patient falls limply down on attempting to stand or walk; and the *ataxic astasia-abasia,* where the patient from the very moment he tries to stand or walk carries out all sorts of jerky involuntary movements, always on the point of falling —on the whole, walking on the floor as if he were balancing on a tight-rope.*

In the distinction of hemiplegic gait from the hysterical " imitation " of it, observation of the *side gait* may be instructive.

The patient is asked to move sideways along a straight line. A patient with pyramidal hemiplegia manages quite well to do this in the direction of the paretic side; but to the non-paretic side he finds it more difficult, and a marked dragging of the paretic leg occurs. In hysterical hemiplegia the side gait is impaired in both directions alike.

The foregoing tests are also very useful in cases of—

SIMULATION.

So are the reflex examination and the electrical examination. Whilst in the ordinary clinical work as simple methods and as few instruments as possible should be

* It has struck me that this latter ataxic form is often quite impervious to treatment (whilst the paralytic form is usually gratifyingly amenable to treatment). I have also seen some cases of ataxic astasia-abasia develop after encephalitis, and therefore believe that this ataxic form of astasia-abasia, in a number of cases at least, is of organic origin.

used, the contrary applies where it has to be decided if the patient is simulating. Here it is a good plan to bring a great many instruments and complicated apparatus into play; this bewilders the patient and helps to unmask his simulation; whilst in an organic case it will make no difference, and a neurotic, as a rule, will feel intensely gratified and enthusiastic at a complicated apparatus being brought into play for his sake. In every individual case the observer has to improvise tests suited to the case. Here are only a few useful tests as examples:

If the dynamometer be compressed as hard as possible, and the pressure repeated at intervals of ten seconds, it will be found that after about twenty to thirty attempts the compression becomes appreciably weaker on account of fatigue. If this reaction of fatigue does not take place after thirty attempts, it may safely be concluded that the patient has not been doing his best at the outset.

If the patient pretends to be unable to move a leg or an arm, it is lifted passively for him, and he is asked to let it sink very slowly "in order to avoid hurting it." Many patients give themselves away by this test. If paralysis be present, the limb will fall down, unless there be pronounced rigidity; if the patient lets the limb sink slowly, it cannot be paralyzed.

If the patient declares himself unable to carry out certain arm movements it is sometimes useful to let him lie face downwards on a narrow couch, the arms hanging down either side, and then to ask him quickly, in terms not used before, to accomplish the movements

he claims not to be able to carry out. Many patients in this unaccustomed position are unable to keep a clear idea of what movements they had decided to feign impossible.*

The patient should always be watched discreetly but closely when he undresses and dresses. Pretend to be in a hurry, and ask him to be quick. When undressing, and particularly when dressing after the examination, simulators will often be caught carrying out movements, which during the examination they feigned paralyzed.

Contractures are tested by prolonged efforts of overcoming the influence of gravity. By extension contracture at the knee-joint, *e.g.*, the patient is placed on a couch with both legs protruding at the foot end in such a way that both knees are well outside the edge of the couch. The one leg is supported at the ankle; the other, where there is alleged contracture, is not. After a few minutes the simulant will let his leg sink under distinct signs of fatigue. In gross organic and hysterical contraction this will not occur for hours.

In case of claudication the attendant, who supports the patient whilst he is walking, is instructed to pass suddenly from the one side to the other. During this a simulated claudication sometimes changes side.

It is sometimes difficult to distinguish between a real organic nerve deafness, hysterical deafness, and simulated deafness. The *cochleo-orbicular reflex* is then useful; a sound such as a motor-horn, blown at a distance of

* In simulated monoplegia brachialis a pronounced dissociation of the function of the latissimus dorsi muscle (as described on p. 62) will generally be found. This, however, may also be found n hysterical paralysis of the arm.

6 feet, will in hysterical and in simulated deafness cause a reflex contraction of the orbicularis palpebrarum, which in organic nerve deafness is lost.

People simulating deafness often pretend to understand by lip-reading. By lip-reading alone it is impossible to distinguish certain consonants from one another —*e.g.*, *p*, *b*, *m*, cannot be distinguished from one another, nor can *t*, *d*, and *n*. A man who is dependent on lip-reading for his understanding is therefore unable to distinguish *mutton* from *button*, *no* from *toe*, *die* from *tie*, *mast* from *past*. If the patient be asked to repeat these words, he cannot avoid making mistakes if he is really deaf. If he is shamming, he will probably make no mistakes.*

ELECTRICAL EXAMINATION.

A large "indifferent" electrode is placed on the back, the sternum, or in the patient's hand; another quite small electrode (about 2 centimetres in diameter) is placed over the nerve or muscle that one wishes to test. This is the "active" electrode. The density of current is here at its maximum, whilst at the indifferent electrode it is generally so small as not to elicit any motor reaction. Both electrodes as well as the patient's skin

* How experimental nystagmus elicited by means of a revolving drum can be utilized for unmasking simulated total blindness has already been mentioned (*cf.* p. 27, footnote). Simulation of unilateral blindness may be unmasked by letting the patient read the test types with such a strong convex or concave glass in front of the other eye that reading with this is rendered impossible. A quick series of tests with various glasses in front of both eyes ought to precede the deciding test, in order to bewilder the patient.

ought to be well moistened with weak saline solution (strength fairly irrelevant). The motor points correspond roughly to the points given in Erb's diagrams (*cf.* Figs. 34 to 39); but as there are individual variations, it is well always to locate them in every case if possible,* and mark their position with a skin-pencil. This ought to be done not only in the part which particularly we wish to test, but also in the symmetrically corresponding part on the other side of the body, as comparisons regarding the strength of current required to produce contraction ought to be made whenever possible.

The strength of the galvanic current can easily be measured by a milliampèremeter; when comparing the galvanic irritation in two different parts of the body a galvanometer reading should always be taken, as the current sent through the body may vary a great deal even if the rheostat be left untouched, this variation being due to the difference in resistance of the skin in different parts, chiefly owing to different degrees of moisture—the resistance also varying in one and the same place, being continually diminished during the flow of the galvanic current. The faradic current cannot easily be measured exactly, but as the voltage is so very high in relation to the resistance offered by the skin, we can take the distance of the two coils as an approximate measure of the stimulus.†

* The motor point is the spot (sometimes a line) where by faradic stimulation the strongest contraction of the muscle in question is elicited.

† This may also be taken as an approximate measure of the stimulus when the faradic current is used for sensory examination, which sometimes it may, in addition to the methods already described under Sensory Examination.

FIGS. 34 TO 39. THE MOTOR POINTS.

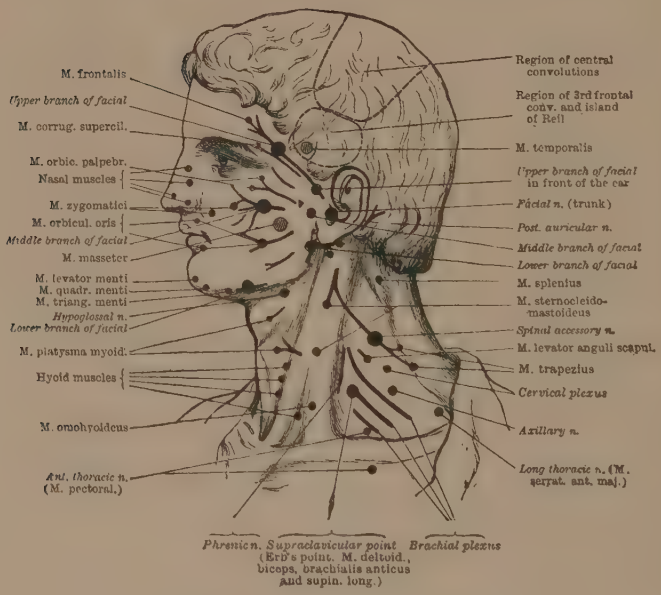

FIG. 34.—THE HEAD AND NECK.

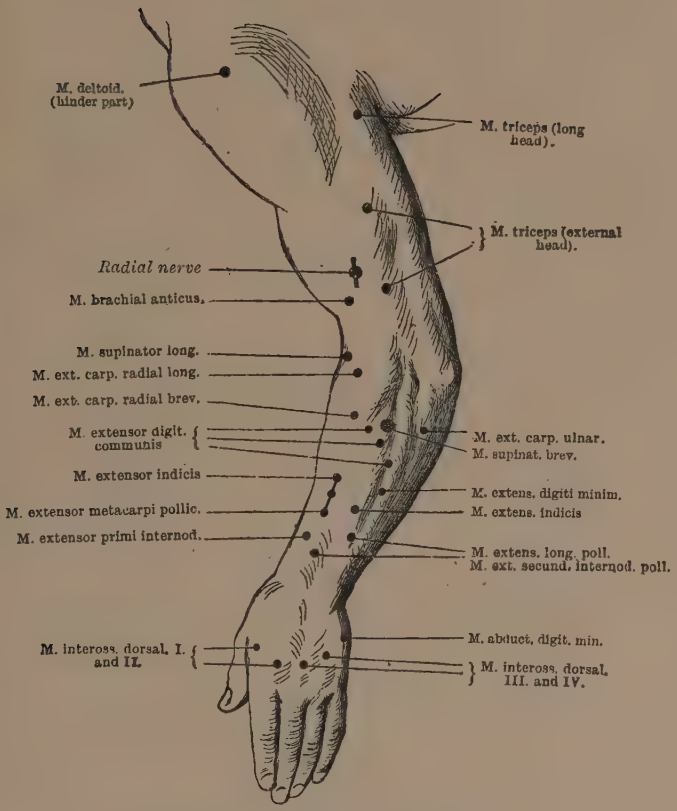
M. deltoid. (hinder part)
M. triceps (long head).
M. triceps (external head).
Radial nerve
M. brachial anticus.
M. supinator long.
M. ext. carp. radial long.
M. ext. carp. radial brev.
M. extensor digit. communis
M. ext. carp. ulnar.
M. supinat. brev.
M. extensor indicis
M. extens. digiti minim.
M. extensor metacarpi pollic.
M. extens. indicis
M. extensor primi internod.
M. extens. long. poll.
M. ext. secund. internod. poll.
M. inteross. dorsal. I. and II.
M. abduct. digit. min.
M. inteross. dorsal. III. and IV.

FIG. 35.—THE UPPER LIMB (BACK).

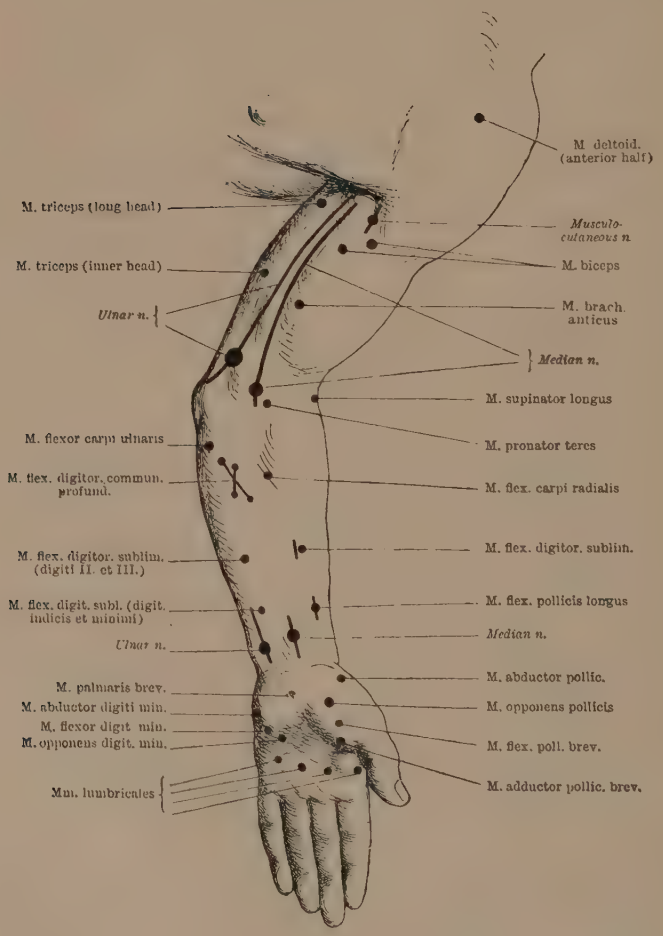

FIG. 36.—THE UPPER LIMB (FRONT).

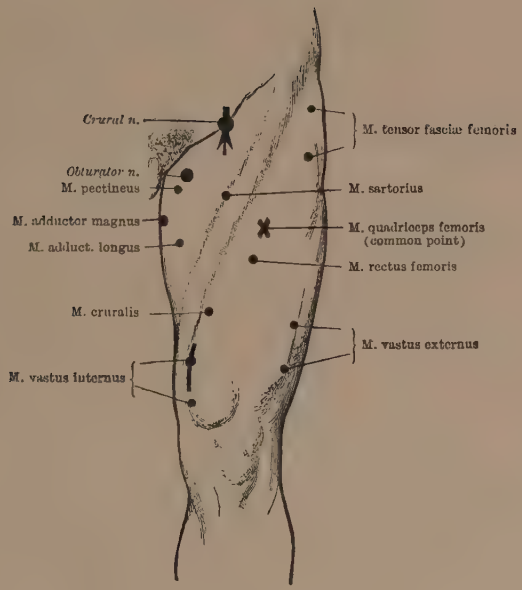

FIG. 37.—THE THIGH (FRONT).

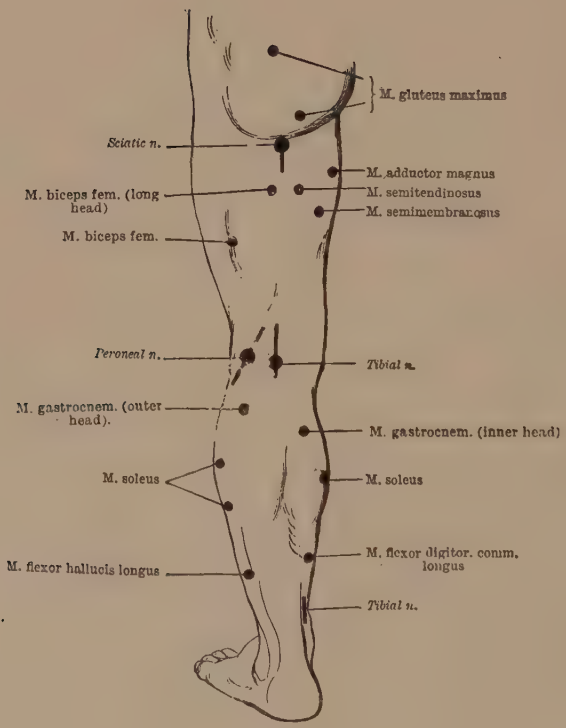

FIG. 38.—THE THIGH AND LEG (BACK).

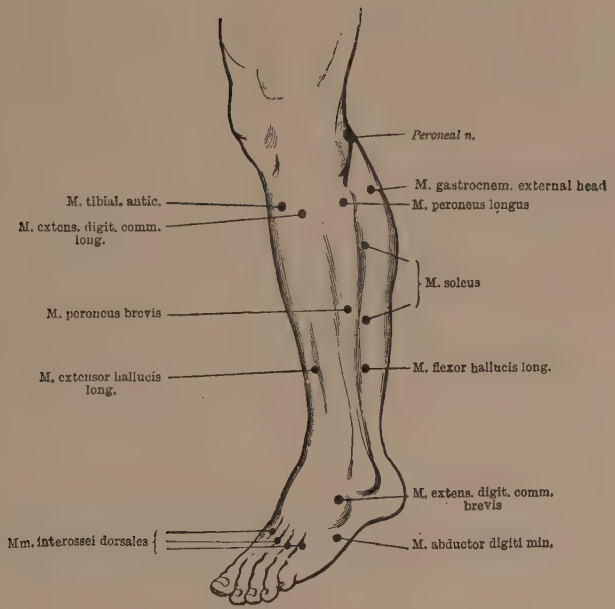

FIG. 39.—THE LEG AND FOOT (OUTSIDE).

Normally faradic irritation of a muscle or nerve produces a tonic contraction,* whilst galvanic irritation only gives a *single, quick, short* contraction at the making (closing) and—if the current be strong enough—at the breaking (opening) of the current. We can either use the anode or kathode as active electrode, and normally we find that there is a certain relation between the different forms of irritation, contractions being most easily elicited on (1) closing the current when the kathode is used as active electrode (kathodic closure contraction =K.C.C.); the other galvanic irritations follow in the order—

(2) Anodic closure contraction (A.C.C.).
(3) Anodic opening contraction (A.O.C.).
(4) Kathodic opening contraction (K.O.C.).

Thus normally we have the formula:

K.C.C.>A.C.C.>A.O.C.>K.O.C.

The two latter (the opening contractions) require, however, such a strong (and painful) current that for practical purposes they are as a rule omitted.

In the ordinary clinical examination we test with the faradic current first, then with the galvanic current. Normally we find a well-maintained tonic contraction on faradic irritation; a single, short contraction (a twitch) on galvanic irritation (K.C.C.>A.C.C.).

* When the frequency of the interruptions is very low, a series of single clonic contractions will be produced; but in ordinary diagnostic routine work one generally uses faradic irritation of such frequency that the single contractions merge into a tonic (or tetanic) one.

When a lesion of the peripheral motor neuron has attained a certain intensity we find—

(1) By nerve irritation, diminished or abolished reaction both to faradic and galvanic stimuli.

(2) By muscle irritation, decreased or abolished reaction to faradic stimuli—a slow, worm-like contraction as response to galvanic irritation, the normal formula (K.C.C.>A.C.C.) being at the same time reversed (K.C.C.<A.C.C.), or at least altered (K.C.C.=A.C.C.).

At the same time, the irritability is found not so strictly limited to the motor points as normally, the whole muscle often showing a distinctly increased irritability to galvanic stimulation.

These alterations of the normal reaction are comprehended by the term *reaction of degeneration* (R.D.) which is always conclusive proof of a *lesion of the peripheral motor neuron.* The most important feature of R.D. is the *slowness* of the contraction by galvanic stimulation.

It has to be borne in mind that the R.D. is not fully developed till a week or two after the onset of the paralysis. Thus, in facial paralysis a reliable electrical examination can first be carried out after a fortnight from the onset of the paralysis.

If the faradic irritability is abolished, we speak of *complete* R.D.; if there is still some reaction to faradic stimulation, it is called *partial* R.D.

The electrical examination is of great prognostic importance.

The prognostic inference to be drawn from the

electrical examination in a case of facial paralysis will be seen from the following table:

Result of Electrical Examination.	*Probable Duration*	*Probable Degree of Recovery.*
Complete R.D.	From three months to a lifetime	Incomplete
Partial R.D.	Six weeks to three months	Complete
Simple diminution of electrical irritability	Two to six weeks	Complete

In *myasthenia gravis* the contractions on continued testing very soon get weaker. The weakening is most pronounced on prolonged faradic irritation, the contracted muscle gradually relaxing in spite of the stimulation being continued. After a short period of rest (varying from case to case) the muscle recovers its contractility again. This phenomenon of peripheral fatigue demonstrated by electrical irritation is called *myasthenic reaction*, and is characteristic of myasthenia gravis. It is most instructive to include the patient and a control individual in the same circuit, and stimulate the same muscle in either by means of electrodes of the same size. In doubtful cases this comparison with a normal individual thus included in the same circuit should always be made. Only the muscles of the limbs should be tested for the myasthenic reaction.

In *tetany* the irritability of the motor nerves to galvanic stimulation is greatly increased. The K.C.C. (kathodic closure contraction) may be elicited by

unusually weak currents; and when the current is increased to about the strength which would normally elicit a single clonic contraction, a *tetanic* contraction is produced. Also the K.O.C. (kathodic opening contraction) appears in tetany on weak galvanic stimulation.

In *myotonia congenita* (Thomsen) a galvanic current will produce a series of slow, worm-like contractions proceeding from the kathode to the anode. With the faradic current the contraction persists for some seconds after the current has been stopped.*

Sometimes a twitch is obtained as K.O.C. as well as K.C.C.; this is called "Rich's reaction." It is reported in muscles paralyzed by pressure on the nerve trunk.

Sometimes on galvanic stimulation a muscle shows a stronger and slower contraction when it is stimulated at its tendinous end than when it is stimulated at its motor point. This is called "longitudinal reaction," and is really only a feature of the reaction of degeneration, which may be more or less pronounced.

The above only represents the chief points in the electrical examination. It requires a certain amount of practice and a great deal of care to make a reliable electrical examination.

(That the electrical stimulation may be utilized in the examination of the sensory system has already been mentioned on page 82. How a weak galvanic current may be used in the examination of taste has

* A similar persistence of the contraction, or rather a retarded relaxation after faradic stimulation, has been observed by Söderbergh in certain extrapyramidal motor lesions, and by him given the name of *myo-dystonic* reaction. This retarded relaxation may be interrupted by one or more clonic contractions.

been described on page 41. Testing the vestibular apparatus by means of a galvanic current has been described on pages 46-47.)

EXAMINATION OF CEREBRO-SPINAL FLUID.

In several hospitals the *lumbar puncture* and the examination of the cerebro-spinal fluid is carried out by the house-physician in every case as part of the routine examination. It is therefore well to mention the one contra-indication to lumbar puncture: cerebral tumour,* and particularly a subtentorial tumour.

Some patients are apt to suffer with headache, sometimes of great intensity, for a few days after the lumbar puncture. This is mostly due to leakage of cerebro-spinal fluid through the puncture opening in the meninges. In order to avoid this leakage it is important that the needle should not be too thick. The outside diameter should not exceed 1·2 millimetre. For this reason also the patient should not be allowed to walk about immediately after the puncture, but should be kept in bed for two or three days. He should lie with his head low, either without or with the smallest possible pillow under his head. The foot end of the bed should be raised. (In the author's clinic the patients are also made to lie face down without a pillow for the first one or two hours after the puncture.)

* Exceptionally lumbar puncture may be carried out even when the diagnosis of *cerebral tumour* has been made. But in these cases—and in all cases where there is a suspicion of cerebral tumour—a *very fine needle* should be used, ensuring a very slow escape of the cerebro-spinal fluid; and the patient should lie with his head very low.

For lumbar puncture the patient may either sit* or preferably lie on his side; in either case he must arch his back as much as possible, trying to make his head and his knees meet. Paint the lumbar sacral region with tincture of iodine, at the same time marking, also with the iodine (if no skin-pencil is at hand), the spinæ ilii.

A transverse line drawn between these indicates approximately the level at which lumbar puncture should be performed. Feel for the processus spinosi, and in the interval between two of these, and about 1 centimetre from the middle line (or, when patient is lying, 1 centimetre *below* the middle line), the needle is pushed through the skin in a forward direction, at the same time slightly inwards and upwards (the direction had better be studied on a skeleton before performing the first lumbar puncture). A slight resistance is felt as the dural sac is pierced, and the fluid trickles out normally drop by drop; if the pressure is increased it escapes in a jet. In adults the needle must, as a rule, be pushed in about 5 to 6 centimetres. It frequently happens that a little blood is mixed with the cerebro-spinal fluid to start with. Cerebro-spinal fluid thus blood-tinged is useless for purposes of examination, and must be discarded. After having let out a few cubic centimetres, however, the blood, as a rule, disappears; then the cerebro-spinal fluid may be collected; but even so an initial admixture of blood should be recorded.

* Pressure reading and pressure gauging are both impossible when the patient sits up. For this and other reasons (danger of unpleasant reaction) the sitting position should only exceptionally be used for lumbar puncture.

By the way the fluid runs out an impression of the intrathecal pressure is obtained. The intrathecal pressure can also be measured manometrically. Normally in the recumbent position it varies between 50 and 150 millimetres of water (according to Eskuchen 40 to 200 millimetres of water). When this is carried out, it must be done before any of the cerebro-spinal fluid has been removed, with the most scrupulous aseptic precautions, and always in the same position—viz., the patient lying on his side with his head as low down as possible.

Whilst the absolute value of the intrathecal pressure is of lesser practical importance,* the *measuring* of *the pressure under the influence of jugular compression* is of the greatest diagnostic importance in cases where subarachnoid obstruction (block) is suspected. For this purpose any glass tube of about 1 millimetre inside diameter may be utilized. It may be connected with the needle by means of a short rubber tube (a few centimetres long). The exact value of the initial reading is of comparatively little interest. The all-important question is: *Does jugular compression cause an immediate rise of pressure or not?* Normally light pressure on both jugular veins causes an immediate rise of intra-

* Every different position of the head gives a different reading, and even the individual width of the shoulders and of the pelvis and the curvature of the spine have some slight influence on the intrathecal pressure as measured by means of lumbar puncture. No great importance can therefore be attributed to small variations, and the big ones will, as a rule, be noticeable enough even without "measuring." *Cf.* also Solomon, Pfeiffer, and Thompson, *American Journal of Medical Science*, September, 1923.

thecal pressure (Queckenstedt).* If this rise does not occur, or even when it is sluggish, this fact indicates that the communication between the intracranial, subarachnoid, and ventricular reservoirs, on the one hand, and the lumbar cistern on the other, is not free.† Only where the intrathecal pressure is very low (subnormal ?), it sometimes happens that the jugular compression causes no rise, even though the communication throughout the whole cerebro-spinal fluid system is free (Ayer).‡ One must take great care that the patient's neck is not previously subjected to any pressure from the clothes. Also the assistant, told off to exert the jugular compression, must be warned against pressing on the larynx, as this can produce a cough and thereby rise of pressure, even where there is complete subarachnoid block (Ayer).

In some individuals pressure on one jugular vein alone is sufficient to produce rise of the intrathecal pressure. According to Tobay and Ayer, in complete sinus thrombosis the lumbar pressure is unaffected by jugular compression on the side of the affected sinus, whereas jugular compression on the unaffected side produces an excessive rise,

* The mechanism is obviously this: pressure on jugular veins ⟶ intracranial venous hyperæmia ⟶ increased intracranial pressure ⟶ increased cb.-fl. pressure.

† Another important sign pointing in the same direction is a rapid decrease of the intrathecal pressure to O after the escape of a small quantity of cb.-fl., and a correspondingly quick decrease in the rate of escaping of the cb.-fl. (It must be ascertained that the point of the needle has not slipped out of the subarachnoid space.)

‡ In some instances, however, when I have failed to obtain a rise on jugular compression this has promptly appeared, when the needle has been pushed in a little farther.

commensurate with the normal reaction observed, when both jugular veins are compressed synchronously.

The normal cerebro-spinal fluid is a clear, colourless fluid, which does not coagulate spontaneously. It has the same osmotic pressure as blood-plasma (Mestrezat). In complete obstruction of the spinal canal (*e.g.*, tumour, pachymeningitis) the cerebro-spinal fluid assumes a yellow colour (xanthochromia) and a more or less marked tendency to spontaneous coagulation, thus assuming a likeness to blood-plasma.*

The greatest importance attaches to the microscopic, chemical, and serologic examination of the cerebro-spinal fluid.

For the routine examination the following tests will be sufficient:

1. Cell count *à la* Fuchs-Rosenthal.
2. Total proteid test (Bisgaard's method).
3. Globulin test—Am_2SO_4.
4. Pandy's test.
5. Test for blood.
6. Permanganate of potassium test.
7. Test for glucose.
8. Quantitative estimation of chlorides.

(9. Wassermann's reaction.)

(10. Colloidal gold reaction.)

* For the diagnosis of meningitis, one may give the patient 2 grammes of *uranin* (sodium fluorescine) by the mouth three hours before lumbar puncture. Only in cases of meningitis the substance is found in the cerebro-spinal fluid, where its presence is easily discovered by mere inspection on account of its fluorescence. (This fluorescence does not disappear in a solution till diluted to 1 in 10,000,000.) *Cf.* O. Jervell: *Acta Medica Scandinavica*, Supplement vii., 1924, p. 115.

The last two methods are generally left to the pathologist to carry out; the other eight should be carried out by the clinician.

1. For the cell count a staining fluid consisting of 5 per cent. solution of acetic acid containing 0·5 per cent. methyl violet is employed. Of this fluid 1 mm.^{3} is drawn up in the pipette (which is the same as used for the leucocyte count in blood) and mixed with 10 mm.^{3} of cerebro-spinal fluid. After being well mixed, a drop of the mixture is placed on the Fuchs-Rosenthal counting-stage. The first drop that comes out must be discarded.

The cells ought to be counted in all the sixteen squares of the counting-stage; the total capacity of all the squares is 3·2 mm.^{3} in all. If, therefore, the number of cells counted be divided by 3·2 and multiplied by $\frac{11}{10}$ (the dilution), the number of cells per cubic millimetre in the cerebro-spinal fluid is obtained. For all practical purposes it will be sufficient simply to divide the total number of cells counted by 3. Normally the number of cells found should not exceed 3 per mm.^{3}

The *type* of cells should also be studied. In *acute* inflammatory conditions chiefly *polymorphonuclear* leucocytes are found; in *chronic* inflammatory conditions the chief type of cell is the *lymphocyte*. Tumour cells are rarely found in the cb.-fl.

2. The *total amount of proteids* in the cerebro-spinal fluid is best estimated by finding out how much the cerebro-spinal fluid has to be diluted before the proteid test becomes negative. The most handy test is the "ring test" with nitric acid; it is only Heller's

test for albuminuria applied to the cerebro-spinal fluid.

The cerebro-spinal fluid, diluted in varying proportions with 0·9 per cent. saline solution, is carefully and slowly allowed to flow on to the surface of the nitric acid. The proteids are precipitated on the border between the two fluids as in albuminuria. (Time limit for reaction, three minutes.)

Normally the test should become negative at a dilution of 1 to 15 or less.

3. The *globulin* is estimated by the ammonium sulphate test. We know that the globulins are precipitated in a semi-saturated solution of Am_2SO_4. Consequently, on mixing equal parts of the cerebro-spinal fluid and a saturated solution of Am_2SO_4 the globulins present will be precipitated and make the mixture more or less cloudy (Nonne and Appelt).

This can also be carried out as a " ring test," allowing the cerebro-spinal fluid to flow carefully and slowly on to the saturated ammonium sulphate solution (Ross and Jones); where the two fluids meet there will be formed a zone of semi-saturated ammonium sulphate solution, and here the globulins will be precipitated in a " ring," which is easily observed between the clear fluid above and below. In order to get an estimation of the quantity of globulins present, the procedure is the same as in the estimation of the total proteids given above—*i.e.*, we repeat the test with cerebro-spinal fluid diluted in varying proportions with water, and note at what ratio of dilution the test becomes negative. (Time limit for reaction, three minutes.)

The nitric acid reaction *à la* Heller is normally found to disappear at a dilution of from 1 in 6 to 1 in 15, whilst the globulin reaction disappears at a dilution of 1 in 2, if it is at all present.

It is doubtful if the substance precipitated from the cerebro-spinal fluid by ammonium sulphate, as described above, is real globulin or globulin only. This, however, does not invalidate the practical value of the test.

Increase in the amount of proteids is chiefly found in inflammatory conditions of the meninges, tabes, and general paralysis; it is regularly accompanied by an increase in the amount of globulins present, and this globulin increase is in general paralysis sometimes out of proportion to the amount of total proteids.

In inflammatory conditions it is also generally accompanied by an increase in cells. When an increase of proteids occurs without a corresponding increase in the number of cells, it is suggestive of an obstruction of the subarachnoidal space (subarachnoid block)—*e.g.*, spinal tumour.

This increase in the amount of proteids without any corresponding increase in the number of cells may be accompanied by xanthochromia (in some cases also a tendency to spontaneous coagulation). These changes are characteristic of the cerebro-spinal fluid *below* the obstruction (Nonne, Froin).

Cushing and Ayer have shown, however, that similar changes (even xanthochromia and spontaneous coagulation) may also be found in the cerebro-spinal fluid *immediately above* a tumour, causing subarachnoidal obstruction. Pressure readings in connection with

jugular compression and the lipiodol X-ray examination will then guard one from error as to focal diagnosis.

A moderate increase of proteids unaccompanied by any increase in the number of cells may also be found in *cerebral tumours* and in *polyneuritis*.

This latter finding makes it probable that the lymph spaces of the peripheral nerves drain into the subarachnoid space. [The greater part of the cerebro-spinal fluid is no doubt produced* by the choroid plexus of the ventricles—*cf.* Dandy's experiments; but it is probable that it receives admixtures from other sources—viz., the lymph spaces of the peripheral nerves and the Virchow-Robin perivascular spaces.]

4. *Pandy's test* is also a "globulin" test in a similar sense. For this test a solution of 1 part carbolic acid (crystal) in 15 parts of distilled water is employed. Just 1 drop of cerebro-spinal fluid is let fall into 2 c.c. of this fluid. A bluish-white clouding constitutes a positive reaction. This test is very sensitive.

5. The test for blood in the cerebro-spinal fluid is best carried out by means of a modification of van Deen's guaiac test devised by my assistant, Dr. Saethre: To ½ c.c. of the usual emulsion of equal parts of old turpentine and tincture of guaiacum (freshly prepared), cerebro-spinal fluid (1 c.c. or a little more—not less)

* Whether the cb.-fl. is to be regarded as a product of secretion or as a dialysate is still an open question, the latter possibility being the more probable hypothesis in the light of our present knowledge.

The *absorption* of the cb.-fl. probably takes place by a simple process of filtration through the arachnoid villi into the venous sinuses, the pressure in these being lower than the pressure of the cb.-fl.

is added, and then four or five drops of acetic acid (25 per cent.).*

The reaction is counted as positive when a blue colour appears within fifteen seconds.

6. In the *permanganate of potassium test* (Boveri) equal parts of cerebro-spinal fluid and 0·1 per mille solution of potassium permanganate are mixed in a test-tube, and compared with equal parts of the same potassium permanganate solution and distilled water mixed in another tube. In inflammatory conditions of the meninges the colour of the solution, containing cerebro-spinal fluid, changes into pale yellow.

7. The normal cerebro-spinal fluid contains *glucose* (about 0·055 per cent.). This is lost or diminished in acute meningitis, and frequently diminished in chronic meningitis, tabes, and general paralysis and, as a rule, increased in epidemic encephalitis (*E. lethargica*). The *qualitative* test is carried out with Haine's solution:

Sulph. cupric. 2·5, glycerin 125, potass. hydrate 10, aqu. dest. ad 500 grammes (see *Journal of American Medical Association*, 1920, vol. i., p. 301).

Three drops of this solution are added to 1 c.c. of cerebro-spinal fluid, which is then heated to boiling. When, as normally, glucose is present, a reddish-yellow precipitate is formed. When the amount of glucose falls below 0·01 per cent. this test becomes negative. Normally the cerebro-spinal fluid contains about 0·055 per cent. glucose. (The limits of normal variations seem to be 0·04 and 0·08 per cent.)

* By thus rendering the fluid acid we obtain the optimal concentration of hydrogen ions, which is necessary for detecting very small amounts of blood (1 : 20,000).

The *quantitative* glucose test is of great importance in the differential diagnosis between epidemic encephalitis (where the amount of glucose is, as a rule, increased—hyperglycorrhachia) and different forms of meningitis (where the amount of glucose is, as a rule, greatly diminished—hypoglycorrhachia). The quantitative test had better be carried out by a trained pathologist, however, and the method will therefore not be described here.* (For the usual methods—Bang's and Hagedorn's—see *Biochem. Zeitschr.*, 1918, vol. lxxxvi., p. 264, and *Ugesskrift for Læger*, 1918, No. 31.)

Investigations in my clinic by Dr. A. Schjött have shown that the amount of glucose in the cerebro-spinal fluid does not show the same variations as in the blood. After oral administration of glucose (one gramme per each kilogramme of body weight) no increase of glucose could be found in the cerebro-spinal fluid within 1½ hours after ingestion. Between 1½ and 2 hours after ingestion an average increase of 0·003 per cent. was found. Between 2 and 3½ hours the average increase was 0·005 per cent.

Only in *epileptics* was the increase of the amount of glucose in the cb.-fl. found to be considerably higher—viz., about 0·015 per cent.

8. A quantitative estimation of the *chlorides* (normally 0·70 to 0·77 per cent.†) may also be useful. A raised

* Borberg gives the following figures: In acute, purulent meningitis, traces of glucose up to 0·013 per cent.; in tubercular meningitis, traces up to 0·042 per cent. (average 0·02 per cent.); in syphilitic meningitis, 0·018 to 0·062 per cent. (average 0·041 per cent.); in dementia paralytica, 0·025 to 0·062 per cent. (average 0·048 per cent.); in tabes, 0·017 to 0·077 per cent. (average 0·051 per cent.).

† The cerebro-spinal fluid is richer in chlorides than blood-plasma, whilst the latter is richer in proteins and a number of other ingredients (uric acid, calcium, sulphates, glucose, cholesterol, inorganic phosphorus). It is the higher chloride contents which gives the cb.-fl. the same osmotic pressure as blood-plasma.

percentage of chlorides is an indication of renal inadequacy; a slightly lowered percentage (0·68 to 0·70 per cent.) is an indication of an acute general infection or of slight meningitis. Figures below 0·68 per cent. are indicative of grave meningeal infection; a percentage below 0·60 per cent. is found in tubercular meningitis only.

Method: 2 c.c. of cb.-fl. are mixed with 10 c.c. of distilled water, a few drops of potassium chromate solution being added as an indicator and a pinch of pure calcium carbonate to ensure alkalinity. Silver nitrate solution (5·814 grammes $AgNO_3$ to the litre) is then added from a burette until the lemon colour changes to orange. Each c.c. of silver nitrate solution used corresponds to one per mille (0·1 per cent.) of chlorides (Buzzard and Greenfield).

(9. The technique of the Bordet-Wassermann reaction cannot be dealt with here. When the question of syphilis arises, it ought to be carried out both in the blood and the cerebro-spinal fluid. But this as well as

10. The *colloidal gold reaction*, the Guillain-Laroche-Lechelle reaction, and the mastix reaction all belong to the domain of the trained pathologist.)

If in spite of the precautions mentioned on p. 136 the patient should develop a headache, some leakage of cerebro-spinal fluid has after all probably taken place. An intravenous injection of 40 c.c. sterile distilled (or preferably redistilled) water may then be tried. By lowering the osmotic tension of the blood it probably increases the formation of cerebro-spinal fluid.*

* On the other hand, an intravenous injection of a hyperosmotic solution will tend to diminish the amount of cb.-fl. (*cf.* pp. 209-210).

Puncture of the Cisterna Magna (inaugurated by Wegeforth, Ayer, and Essick) is another method of obtaining cerebro-spinal fluid. It cannot be recommended as a routine method replacing lumbar puncture, but is nevertheless a valuable method, particularly for the injection of lipiodol for radiographic purposes (Sicard). The patient should be lying on his side, the head bent forward without any rotation or any flexion to either side. The spine of the axis (epistropheus) is easily found and the needle, which should be 6 centimetres long, and armoured with a well-fitting stylet, should be pushed in just above, exactly in the middle line, in a direction roughly indicated by a line through the external auditory meatus to the glabella. Preferably, however, the needle should be directed in a slightly more cephalic direction; the danger of damaging the medulla oblongata is thus diminished. As a rule, the needle has to be pushed in about 4 or 5 centimetres before the cisterna is reached; but it should always be kept in mind that, particularly in very thin individuals, the cisterna magna is not always so far removed from the surface. (Trying the method on cadavers, I found that the needle once reached the cisterna at a depth of 2·7 centimetres, in another instance at 3·1 centimetres; the average distance at which the cisterna has been reached in all my punctures, in cadavers and in patients, has been 4·3 centimetres.)* When the needle has been pushed in to a depth of 2·5-3 centimetres, the stylet is removed

* *Cf.* Monrad-Krohn: "Om cisternepunktion," *Norsk Magazin for Lægevidenskaben*, August, 1923, No. 8, p. 711.

to see if any fluid escapes; if not, the stylet is replaced, and the needle pushed in another 0·5 centimetre, the stylet then removed again. In this way one should proceed by half centimetres till fluid is obtained from the cisterna magna. The needle should, however, on no account be pushed in to a depth exceeding 6 centimetres.

In doubtful cases of subarachnoid block (*e.g.*, spinal tumour), it is a great advantage to be able to compare the fluid obtained by lumbar puncture with that obtained by puncture of the cisterna magna; also the comparison of the pressure in the lumbar cistern and the pressure in the cisterna magna may be very instructive; and finally, puncture of the cisterna magna is *the* method for injecting lipiodol ("descending lipiodol" Lafay, 54 per cent.*) into the spinal canal for radiographic purposes. The lipiodol injected into the cisterna will sink down to the lowest part of the subarachnoid space (the sacral cul-de-sac) unless it meets with an obstacle, which arrests it at a higher level. This can easily be controlled by a series of X-ray photographs of the spinal column. The patient should be sitting, not lying down,

* Lately an "ascending lipiodol" (Lafay, 11 per cent.) has also been introduced for lumbar injection; it may be useful both for verifying the diagnosis of subarachnoid obstruction and for determining the *lower* border of such obstruction; but it does not give such a deep shadow in the radiogram as the descending lipiodol (54 per cent.) does (*cf.* Fig. 40). Personally I have a fear that it may cause obstruction of arachnoidal villi (which again might cause hydrocephalus), and therefore I do not like to use it except for determining the lower border of a complete obstruction which has already been shown by the descending lipiodol (*cf.* Fig. 40).

when the photographs are taken; he should sit up immediately after the injection has been made, and remain sitting until photographed.

The sacral cul-de-sac is so situated that the lower end of the lipiodol shadow normally is found at the level of the first to second sacral vertebræ (when photographed in standing or sitting position: *cf.* Fig. 41).

The descent of the lipiodol may also be observed on the *screen* immediately after the injection. Thus an *incomplete* obstruction may also be detected and the observer is enabled to take a series of X-ray photographs in quick succession, whilst the lipiodol passes the obstruction.*

The cisterna magna route should also be *the* method for subarachnoid administration of medicamina, being the most accessible centre of distribution. Medicamina, injected into the cisterna magna, will reach both the spinal cord and the convexity of both hemispheres.

* Readers interested in the diagnostic (and therapeutic) use of lipiodol are referred to " Diagnostic et thérapeutique par le Lipiodol," by J. A. Sicard and J. Forestier, Paris, 1928.

FIG. 40.—SUBARACHNOID BLOCK (TUMOUR).

Located between the deep upper-shadow of the descending lipiodol (injected into the cisterna magna) and the less saturated shadow of the ascending lipiodol below (injected by the lumbar route).

To face page 150.

FIG. 41.—LIPIODOL SHADOW IN THE SACRAL CUL-DE-SAC UNDER NORMAL CONDITIONS.

To face p. 150.

APPENDIX

I. MEASUREMENT OF INTELLIGENCE.

(*The Binet-Simon System of Tests.*)

In order to get some kind of "measurement" of intelligence, various authors have devised several standard tests, by means of which it is possible to get some quantitative and comparable data of the intelligence of a patient. The most important of these tests are those devised by Binet and Simon. The Binet-Simon system contains a great number of tests, arranged in groups so as to correspond to the various stages of development of the normal individual. By means of these tests one can determine the "intellectual age" of the individual, and this will, in mentally defective individuals, often be found to differ considerably from their real age—*e.g.*, a mentally defective boy of nine years may only reach the fifth-year standard.

No two individuals develop exactly along the same lines—one individual is ahead of the average standard in one respect, behind in another; therefore the different standards are necessarily somewhat overlapping. To determine the "intellectual age" of an individual exactly is very difficult. The correct evaluation of the different answers to the various tests is likewise very difficult, and requires great practice. In the following only an outline of the Binet-Simon system and the most

important of its tests can be given.* After the tests that also belong to another standard than that under which they are given, a figure in brackets gives this other standard.

Third Year.—Point to the mouth, the eye, the nose. Repeat sentences consisting of six syllables or two digits. Indication of the different things seen in a picture. Surname.

Fourth Year.—Name different familiar objects shown (key, knife, watch, pencil). Repeat three digits. "Are you a boy or a girl?" Comparison of two straight lines of different length (5 and 6 centimetres).

Fifth Year.—Repeat sentences of ten syllables. Count four pennies. Copy a drawing of a square. Compare two weights (3 and 10 grammes). Form a rectangle from two triangular pieces of cardboard . Count four pennies.

Sixth Year.—Definition of concrete objects by naming their use—*e.g.*, pencil, carriage, key, etc. ("What is a key?" "To open a door with.") Simple æsthetic comparisons. (An exceedingly ugly and a handsome face are drawn, and the child is asked which is prettier [Fig. 42].) Distinction of right and left (7). Distinction between morning and afternoon. Fulfil three simple requests made simultaneously—*e.g.*, "Put this book on the table," "Shut the door," and "Bring me that pencil" (7). Repeat sentence of sixteen syllables. Copy the

* For details regarding measurements of intelligence, see L. M. Terman: "The Measurement of Intelligence," London, 1919, and Cyril Burt: "Mental and Scholastic Tests," London, 1922; and Drummond's translation of Binet and Simon's "Mentally Defective Children."

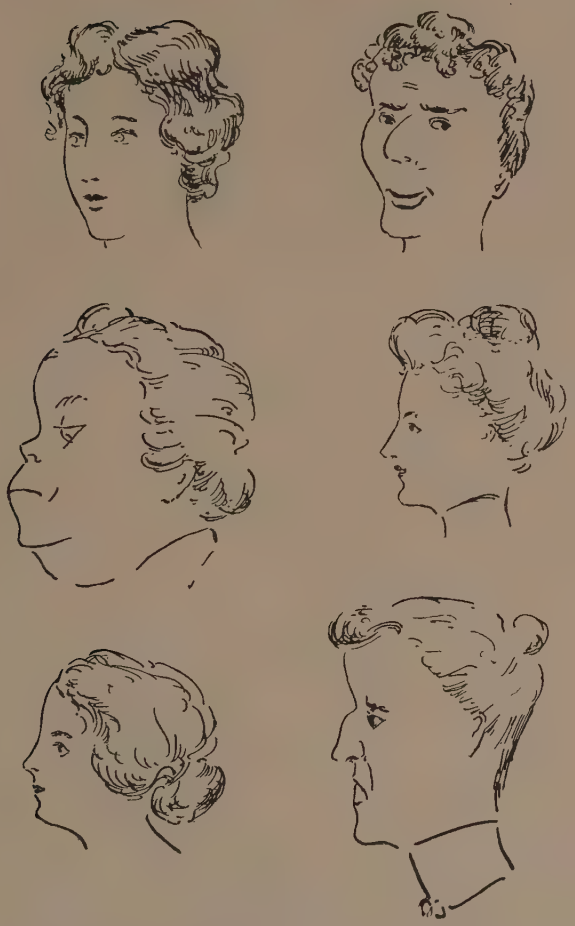

FIG. 42.

From Dr. Drummond's translation of "Mentally Defective Children," by Binet and Simon (Arnold, London).

drawing of a lozenge-shaped figure (7). Count thirteen pennies (7).

Seventh Year.—Description of simple objects—*e.g.*, table, chair. Fulfil three simple requests made simultaneously (6). Distinction of right and left (6). Collect ninepence from different coins—penny and halfpenny pieces (8). Name the four chief colours (red, blue, green, yellow).

Eighth Year.—Count backwards from 20 to 1. Compare two objects from memory only. What is the difference between a dog and a bird? between a ship and carriage? fly and butterfly? Repeat five digits (7). Discovery of omissions in simple drawings (Fig. 43), (7). Give the date (9).

Ninth Year.—Fuller definition of objects than required under sixth-year standard—viz., not only their use, also what they are made of; *e.g.*, "What is a fence?" "It is something made of wood, stone, or iron to show where the garden or the estate ends." Pay ninepence out of a shilling. The names of the months (10). Name the different pieces of money. Light questions of intelligence, chiefly of a practical kind—*e.g.*, "What would you do if you suddenly discovered that the house was on fire?" "Save people and valuables, walk out, inform fire station," would be the *ideal* reply.

Tenth Year.—Include three given words in two sentences. More difficult questions of intelligence—*e.g.*, "Why should you judge a man by his acts rather than by his words?" Arrange five weights in order of their size—3, 6, 9, 12, 15 grammes (9). Criticism of absurd sentences (11)—*e.g.*, "Why should we not

FIG. 43.

From Dr. Drummond's translation of "Mentally Defective Children," by Binet and Simon (Arnold, London).

bathe in July?" "Why are all cats grey?" "Why does the river run uphill?" etc. Copying of drawings from memory.

Eleventh Year.—Include three given words in one sentence (12). Definition of abstract terms such as justice, truth, kindness (12). Rearrange a number of words into a proper sentence (12). Mention sixty words in three minutes (12). Criticism of absurd sentences (10).

Twelfth Year.—Include three given words in one sentence (11). Definition of abstract terms (11). Rearrange a number of words into a proper sentence (11). Mention sixty different words in three minutes (11). Resistance against linear suggestion.*

Thirteenth Year.—Cut out a certain figure in cardboard. Distinction of abstract terms: What is the difference between lie and misstatement? between killing and murdering?

Fifteenth Year.—Description and interpretation of pictures, not only mentioning the different objects as required already in the third-year standard (12). Repeat seven digits (12). Repeat sentences of twenty-six syllables (12). Make three rhymes in one minute (12). Fill in omissions in certain sentences when

* Technique: six pairs of horizontal lines are drawn on separate sheets of paper. In the three first pairs the line to the right is a little longer than the left, the difference being just big enough to be easily noticeable; in the three last pairs the lines are equally long. When the three first pairs have been shown, and the child has correctly indicated the difference in the length of the lines there is a tendency for the child to say that also in the three last pairs the right line is longer. The suggestion is considered to be resisted when two of the three last pairs are recognized to be equal.

several words have been left out; *cf.* Ebbinghaus's test, p. 6 (12).

Sixteenth Year.—Review the main ideas of a short tale.

It is easily seen that the above indications allow a fair amount of latitude. It will not rarely be found that the child's "intellectual age" is difficult to fix exactly—*e.g.*, one child may fail in certain tests belonging to the sixth-year standard, and yet quite well manage tests belonging to the seventh-year or even eighth-year standard.* The reader is therefore warned against a too rigid application of these tests.

The relation of the "intellectual age" to the individual's real age is called *Intelligence Quotient* (I. Q.).

$$\text{I. Q.} = \frac{\text{Intellectual Age}}{\text{Real Age}}.$$

According to Terman we have the following classification:†

I. Q. above 140—"Near" genius.
,, 120-140—Very superior intelligence.
,, 110-120—Superior intelligence.
,, 90-110—Average intelligence.
,, 80-90—Dull.
,, 70-80—Borderline deficiency.
,, below 70—Definite feeble-mindedness.

Below 70 we can classify thus:

I. Q. 0-25—Idiots.
,, 25-50—Imbeciles.
,, 50-70—Morons.

* The single tests have also been grouped together in different ways (*e.g.*, the Stanford revision—*cf.* Lewis M. Terman: "Measurement of Intelligence").

† In calculating the I. Q. of an adult it is necessary to disregard the years he has lived beyond the point where intelligence attains its final development. Terman assumes this final development to have been attained at the age of sixteen years.

Other standard tests have been devised by Burt. His "reasoning tests" are very valuable for the picking out of particularly gifted children; but for the "measuring" of mental backwardness, the Binet-Simon system remains *the* method.

According to Goddard, the practical ability (the ability of being taught certain practical work) gives a reliable guide for the judgment of the intelligence; a rough scale of "intellectual ages," based on this practical ability, follows:

Age of 1 year:	The individual will eat anything.
,, 2 years:	Distinguishes between eatable and non-eatable things.
,, 3 ,,	Plays a little. No work.
,, 4 ,,	Will try to help others in their work.
,, 5 ,,	Carries out very simple pieces of work.
,, 6 ,,	Works in short spells—*e.g.*, can wash up
,, 7 ,,	Carries out small errands in the house—*e.g.*, can brush clothes and carpet.
,, 8 ,,	Errands in the neighbourhood. Light work—*e.g.*, can make beds.
,, 9 ,,	Heavier work—*e.g.*, scrubbing, bricklaying.
,, 10 ,,	Routine work. Helpful in institutional life under supervision.
,, 11 ,,	More complicated work under supervision.
,, 12 ,,	Routine work without supervision. Can take care of animals. Yet no planning ability.

N.B.—Useful information regarding the intelligence of school children will easily be obtained from intelligent schoolmasters.

II. PSYCHOSOMATIC EXAMINATION

(Bearing upon the Psychosensory and Psychomotor Functions).

The chapter of aphasia has always been one full of unpleasant complications to the student. Simple as this disturbance appears in many textbooks, the first case that he observes is apt to confuse (and depress) him with its complexity. The routine examination will easily disclose if there is a marked disturbance of speech; it will as a rule also enable one to distinguish between an articulatory speech disturbance (dysarthria) and a non-articulatory speech disturbance (aphasia). Judging by the way the patient carries out the numerous requests that are made to him during the routine examination, it may also be possible to distinguish between a disturbance of motor speech (motor aphasia) and a disturbance of sensory speech—viz., perception of spoken language (sensory aphasia).

But as the aim of our clinical investigation is to discover not only part of, but *the whole* functional loss (a *conditio sine qua non* for a rational re-education treatment), a complete examination of all psychosensory and psychomotor functions ought to be made in all cases of aphasia or any other psychosomatic disturbance. In the following an outline of the complete psychosomatic examination is given.

On account of the complex nature of the functions concerned, the author has found it impossible to avoid some introductory considerations before giving the bare technique of the examination.

It has to be borne in mind that every one of our clinical tests really is a double test, consisting of two different components:

(1) The perception of the sensory stimulus—a psychosensory function; and

(2) The motor response—a psychomotor function.

When, *e.g.*, we ask a patient of ordinary intelligence how old he is, and no answer is received, the reason for this may either be that the man does not understand or that he cannot speak. Which is the real reason can only be determined by other complementary tests. When, again, a patient is asked to lift up his left arm and he fails to do so, the reason may either be that he does not understand or that he is unable to perform the movement. In other words, the cause of the defect may be either on the side of the motor or on the side of the sensory functions involved.

It is only through the psychomotor reactions that we gain information about the psychosensory processes, and the psychosensory functions are, on the other hand, involved in all our tests for the psychomotor functions, as necessary for the transmission of the test stimulus.

Thus all our tests comprise one psychosensory and one psychomotor reaction, and it is only by a combination of tests that we can localize the defect to a distinct function.*

E.g., a man is asked how old he is—no response; he is asked to lift his left arm up—no response; he is asked to write his address—no response. Now he is asked in writing to say his name—correct response;

* No *anatomical* localization is meant here.

to lift his left arm—correct response; to write his address—correct response.

From this we are entitled to draw the conclusion that his aural perception of spoken language is deficient, and that this is to blame for the lack of response in the first three tests.

It is now easy to see how we can evolve a complete system of tests, and also how a purely clinical nomenclature can be established on the base of this system of investigation.

It is well to enumerate first the different psychosensory and psychomotor functions we are interested in clinically, and for the sake of an easy survey they have here been tabulated.

Psychosensory Functions.

1. **Aural Perception.**—Disturbance: total auditory agnosia.

(*a*) *Of Words.*—Disturbance: sensory aphasia.

(*b*) *Of Inarticulate Sounds.*—Disturbance: partial auditory agnosia.

2. **Visual Perception.** — Disturbance: total visual agnosia.

(*a*) *Of Words.*—Disturbance: alexia.

(*b*) *Of Drawings.*—Disturbance: "asymbolia."

(*c*) *Of Objects.*—Disturbance: partial visual agnosia.

3. **Tactile Perception.**—Disturbance: astereognosis.

4. **Olfactory Perception.** — Disturbance: olfactory agnosia.

5. **Gustatory Perception.** — Disturbance: gustatory agnosia.

(The last two are of comparatively little importance in the ordinary clinical examination of psychosensory functions, as the psychic functions connected with these sensory functions in man are very scanty compared with (1), (2), and (3).)

Psychomotor Functions.

There are only three ways in which man can give expression to his thoughts (*cf.* Kant's "facultas signatrix.")

I. By means of spoken language (disturbance: motor aphasia).

II. By means of written language (disturbance: agraphia).

III. By means of mimicry, demonstration, and actions (disturbance: apraxia).

Consequently, the psychomotor reaction by which the patient responds to our different tests can only take one of three forms:

I. Spoken response.

II. Written response.

III. "Practical" response, in the shape of actions, demonstrations, and mimicry. (Response by means of detached letters formed into words by the patient may be classed under II.)

In order to have a complete system of examination for all these functions, we have to couple each psychosensory reaction with each of the psychomotor reactions. Simple as this may appear at first sight, it will be found that the whole system, in order to be complete, will comprise a very large number of different tests.

The order and sequence of the different tests can, of course, be varied from case to case, and so can the actual carrying out of the individual tests. The main thing is that the whole system of tests is gone through in its completeness. To facilitate the orientation of the different combinations of psychosensory and psychomotor functions, the diagram on p. 164 may be found useful.

In order not to make the diagram too complicated, the olfactory and gustatory forms of perception are left out.

Perhaps it is as well to point out that this diagram is not meant to have any anatomical significance. Nothing has barred the progress of research in aphasia more than the unwarranted confusion of functions with anatomical centres. It is questionable if centres proper—in the same sense as the centres for different simple movements in the motor area—exist for these complicated psychosensory and psychomotor functions, whose paths necessarily must extend over wide parts of the brain.*

As regards the practical carrying out of this system of tests, we may find it advisable to alter the order of

* The author wishes by no means to underrate the valuable work on the anatomical substratum of these functions done by Broca, Wernicke, Kussmaul, Pierre Marie, Déjerine, Henschen, and others; he only wishes the student clearly to realize that nothing can be gained by an untimely anticipation of an anatomo-clinical correspondence, which no doubt exists, but the details of which still for the greater part remain unknown. Until the necessary knowledge has been gathered, all we can do is to avoid muddling the problems in question—above all, to employ a uniform and exhaustive clinical examination, and to record the results in purely clinical (*i.e.*, functional) terms. *Cf.* the author's article in *Journal of Mental Science*, 1917.

PSYCHOSENSORY FUNCTIONS.
PSYCHOMOTOR FUNCTIONS.
1
AUDITORY PERCEPTION
(a) Of words
(b) Of inarticulate sounds
2
VISUAL PERCEPTION
(a) Of words
(written)
(printed)
(b) Of pictures
(c) Of objects
3
TACTILE PERCEPTION
I
SPOKEN LANGUAGE
II
WRITTEN LANGUAGE
III
ACTIONS AND DEMONSTRATIONS

FIG. 44.

the tests to suit the different cases. The author has found it practical in most cases to proceed in the following order: First, all the different tests are carried out in which the patient responds with spoken language (I.); then those tests where he responds in written language (II.); and finally those where his response takes the form of actions, mimicry, or demonstrations—"practical" response (III.).

Both the examiner's questions and the patient's answers should be accurately recorded (*verbatim*).

I. Perception as Evidenced by Spoken Response.

During the tests under this heading it is necessary not only to record accurately all the patient's answers, but also to note if there is any retardation. Any dysarthria, any deficient phonation, the use of wrong words (*paraphasia*) and of distorted words (*e.g.*, "contipution" for "constitution"—*jargonaphasia*) must be noted. Signs of *perseveration* should also be carefully looked for. This phenomenon is of common occurrence in aphasic disturbances: thus, when enumerating the months the patient may stick at one name—*e.g.*, June—and, however hard he tries to get on to the other months, the name "June" is the only one that comes to his lips—nay, he may even to a series of subsequent questions only be able to give the one answer, "June."

1. **Aural Perception**—(*a*) *Of Words.*—"How old are you?" "What is your name?" "What way did you come here?" "Tell me the days of the week." "Enumerate the months—the alphabet." "Count from 1 to 100, and from 20 backwards to 1." "Recite the first

verse of 'God save the King' and 'Rule Britannia!'" "Sing these songs." "Hum them without words." Arithmetical questions, $2+2$, $6+7$, 2×3, 3×4, 4×5, 5×6, $8\div5$, $26:4$, etc.

(*b*) *Of Inarticulate Sounds.*—The patient is asked to shut his eyes and tell what he hears: rattling of keys crackling of paper, striking of a match, pouring out of water, whistling, imitation of various animals (dog, cat, etc.). The patient may also here be asked if he can recognize a melody played on the piano, etc.

2. **Visual Perception**—(*a*) *Of Words.*—(It is necessary, of course, to be sure that the patient has learnt to read.) Various questions are written for him, and he is asked to answer them verbally *without* reading out the questions aloud:* "When were you born?" etc. He is asked to relate in his own words the contents of some printed matter†—*e.g.*, a short story.

(*b*) *Of Pictures.*—He is shown different pictures, and asked to explain them. We can also draw a simple sketch for him, and note in what stage of the unfinished sketch the patient can "diagnose" it.

(*c*) *Of Objects.*—He is shown different objects (pencil, cigar, book, matchbox, inkstand, lamp, etc.), and asked to name them. He is shown a watch and asked the time.

3. **Tactile Perception.**—The patient is told to shut his eyes, and to name objects placed in either of his hands—*e.g.*, coin, pencil, key (not keys that rattle)

* If he fails, he is allowed to try again by reading the questions aloud before answering them. If he answers the questions satisfactorily, the words should be written vertically, thus making the reading more difficult.

† This ought to be presented to him upside down. Note how long it takes before he turns it round.

(auditory perception). Compare the test of the stereognostic sense (*cf.* p. 87).

4. **Olfactory Perception.**—To name smells applied to his nostrils. Compare the ordinary neurological test of the first cranial nerve.

5. **Gustatory Perception.**—To name tastes applied to his tongue. Compare the ordinary neurological test for taste.

II. Perception as Evidenced by Written Response.

Here the patient is asked to *answer in writing*, if necessary with the left hand,* to the same or similar questions as under I.

III. Perception as Evidenced by "Practical" Response (Actions, Demonstrations, and Mimicry).

As will be readily seen, the "practical" response often takes place in answer to perception through two or three different senses (*e.g.*, groups 3, 4, and 5).

1. **Aural Perception.**—

"Lift your left hand up."	Actions of graduated complication, without object (later, under 3, *with* object).
"Touch your left ear with your right hand."	
"Button your coat."	
"Beckon to a person; show how you would use a key."†	
"Look angry" (mimicry), etc.	
"Whistle a tune."	

* Right hemiplegia is a frequent complication in motor aphasia and agraphia. *Cardboard letters* may also be used with advantage. It is very important to ascertain if the patient can express himself by means of these.

† These tests ought to be carried out with each hand separately. Apraxia may well be confined to *one* limb—"Glied kinetische" apraxia of the Germans (Liepmann).

2. **Visual Perception.**—The patient is asked to carry out written requests of the same kind as mentioned under (1).

3. **Auditory and Visual Perception.**—"Point to a chair, a table, a window," etc. "Take a pencil and draw a house." "You see these three pieces of paper on the table in front of you—the biggest you are to tear up, the smallest you put in your pocket, and the remaining one you give to me" (P. Marie's "Epreuve des trois papiers"). "Go to the window, open it, then knock at the door and come and sit down again."

4. **Auditory, Tactile, and Visual Perception.**—"How do you use this?"—a key, a brush, a tape-measure, etc. Strike a match, etc.

5. **Auditory and Tactile Perception.**—Same as 3, only with eyes shut.

In addition to this system the patient is asked to—

Repeat words. Copy writing.	Automatic actions without any psychic content.
Transcribe printed matter into hand-written language, and *vice versa*. Read aloud.* Write to dictation.	Semi-automatic actions.

Besides, in every case the *spontaneous language* is to be closely observed, particularly with a view to any possible difference between intellectual and emotional

* If he cannot read aloud, he ought to be asked to spell aloud. Can he join the letters to syllables and the syllables to words? If he cannot even spell aloud, he is asked to read single letters and ciphers.

language (more or less highly associated speech), which is so frequently observed. Make a special note of the patient's use of swear words and idiomatic expressions.

In some cases the psychosomatic examination is rendered exceedingly difficult—partly impossible—by incessant *logorrhea* and *jargonaphasia*, which may even make all the patient's language completely unintelligible.

From a research point of view, it is obvious that in educated persons who speak several languages, the examination should be carried out in two or three languages.

The whole psychosomatic examination can only quite exceptionally be completed at one sitting. As a rule it is a work of several days.

On account of the variability of so many aphasic patients to tests (as already pointed out by Hughlings Jackson), every patient ought to be examined repeatedly.

The nomenclature of the psychosomatic disturbances is so arbitrary and partly inconsequential that no observer should content himself by simply fixing one of the ordinary labels ("motor aphasia," "sensory aphasia," "alexia," "agraphia," "apraxia") to the case observed, but *describe* the case as fully as possible based on the complete psychosomatic examination, as here outlined.

It should be recorded whether the patient is right-handed or left-handed.

III. ON DIPLOPIA.

Diplopia will as a rule be discovered readily enough by the simple proceeding described on pp. 24 and 29.

For the sake of a more exact examination and a completer record of the findings, we may advantageously proceed in the following manner:

The patient is placed facing a blackboard, preferably at a distance of 1·15 metres,* and made to hold a red glass in front of his right eye. The observer holds a lighted candle immediately in front of the blackboard, moving it upwards and downwards to the right and to the left, both horizontally and diagonally (about 30-50 centimetres in each direction), the patient being asked to follow the light with his eyes. Where diplopia occurs the patient points out the relative position of the two images, which the observer then immediately marks on the blackboard with chalk. Finally, the marks on the blackboard are copied on to a piece of paper and recorded, as shown in the diagrams on p. 171 (Figs. 45 and 46).

Description of FIGS. 45 *and* 46.

O indicates undisturbed fusion of both eyes. The central O should be approximately opposite the patient's nasion.

\+ indicates the red image of the right eye.

| indicates the uncoloured image of the left eye.

These diagrams, which are based on Professor Schiøtz's numerous observations, give the double images as they are marked on the blackboard according to the method just described—*i.e.*, as viewed from the patient's position. All the other eye muscles are innervated by the third nerve, and therefore rarely affected by an isolated paralysis.

* At this distance a line of 2 centimetres on the blackboard will indicate an angle of 1°.

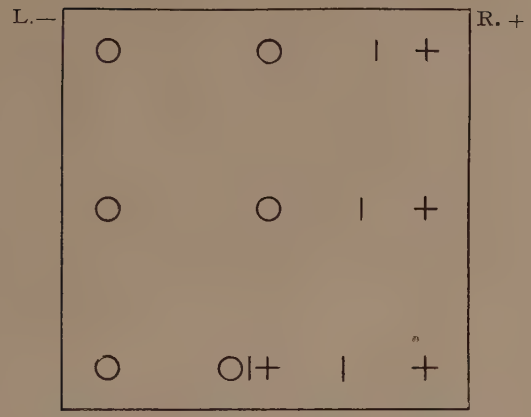

FIG. 45.—PARALYSIS OF RIGHT EXTERNAL RECTUS (PARALYSIS OF RIGHT ABDUCENS).

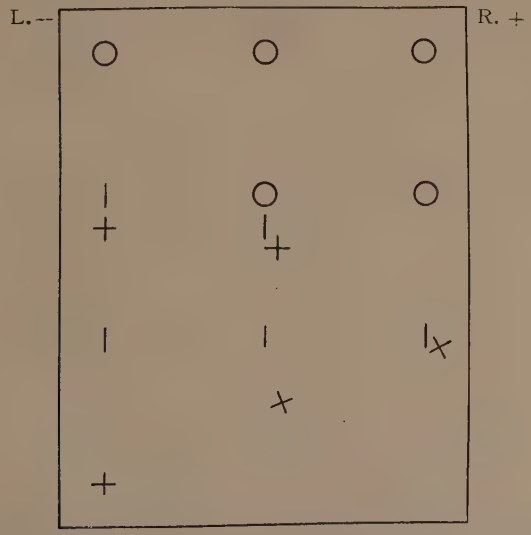

FIG. 46.—PARALYSIS OF RIGHT SUPERIOR OBLIQUE (PARALYSIS OF RIGHT TROCHLEAR NERVE).

For description of these figures see opposite.

It will be found that in paralysis of the fourth (trochlear) nerve the diplopia will be most easily discovered when the patient is looking downward and to the opposite side of the lesion (as indicated in diagram on p. 171). The images are then at some appreciable distance from each other, the one image being above the other.

This seems to the beginner astonishing and out of harmony with the diagram of the isolated action of the various eye muscles given on p. 29.

It has to be borne in mind, however, that the eye movements are not the result of the isolated action of one single muscle, but of the co-ordinated action of *all* the different eye muscles. When the eye is turned about 50° inwards (*i.e.*, in the nasal direction), the superior oblique will be practically solely responsible for the downward movement (the rectus inferior in this position being chiefly a rotator). Thus, in trochlear paralysis the downward movement, in this position of adduction, is abolished, with the result represented in the diagram above as regards the diplopia. When the eye is turned outward the muscle acts as a pure rotator.*

The same considerations also apply to the recti superior and inferior, which are responsible for the vertical movements when the eye is turned about 25° in the temporal direction; and chiefly rotators, when the eye is turned some 50° inwards.

* Paralysis of the fourth (trochlear) nerve is generally regarded as a very rare occurrence, and it is obvious enough that it is not nearly as common as paralysis of the third and sixth cranial nerves. Nevertheless, the author is of opinion that it would be found more frequently if systematically looked for. As regards the anatomy of the trochlear nerve, it is well to remember that curving round the brain stem it comes into the closest proximity of the superior cerebellar peduncles.

IV. VESTIBULAR TESTS.

As these tests are of increasing importance to the neurologist as well as to the otologist, the short technical indications mentioned under the examination of the eighth cranial nerve, are here supplemented with some additional and more general remarks regarding the rotation and caloric tests, which Barany has developed to tests of great practical value.

Before performing these tests, it should always be ascertained if there be any spontaneous nystagmus and involuntary deviation, as this will have to be taken into account when considering the experimental nystagmus and deviation elicited by the vestibular tests. Care must always be taken to distinguish between—

Spontaneous nystagmus,
,, kinetic deviation (" past-pointing "),
,, postural deviation, and
Experimental nystagmus,
,, kinetic deviation (" past-pointing "),
,, postural deviation.

If spontaneous nystagmus is present at certain degrees of deviation, we have to examine for experimental nystagmus in a lesser degree of deviation. A " Blick-fixateur " (Barany)—a rod fixed to the patient's head in such a way as to furnish a point of fixation which can be moved sideways, the amount of deviation at the same time being indicated in degrees—may be of use here, but is not absolutely needed.

In order to exclude such factors which may influence

the nystagmus—*e.g.*, accommodation—it is advantageous, though not necessary, to employ strong convex glasses (Bartel's glasses), thus preventing the patient from fixing any particular object with his eyes. They do not prevent the patient being made to look in a definite direction by the "Blick-fixateur." On the other hand, these glasses act as magnifying glasses to the observer, and facilitate his observation of the nystagmus.

The past-pointing and the postural deviation must from the very start be tested with closed eyes only, as the visual control will at once introduce the bewildering factor of compensation, which is so readily mobilized in all vestibular, and particularly cerebellar, disturbances.

Nystagmus, as well as past-pointing and postural deviation, if vestibular in origin, all conform to the *law of vestibular reaction—i.e.*, the response is determined by the position of the head; *e.g.*, a tendency to fall to the right will, if vestibular in origin, on rotation of the head to the right *ad maximum*, be changed into a tendency to fall backwards. Kinetic and postural deviation always occur in the opposite direction of that of the nystagmus present.

The experimental vestibular nystagmus always occurs in the plane of the semicircular canals which are being stimulated.

The semicircular canals are arranged in three planes, as will easily be seen from the accompanying Ewald's diagram (Fig. 47, p. 175).

It will be noted that each semicircular canal roughly lies in the same plane as one of the semicircular canals on the other side (plane 1 on the left corresponding to

plane 2 on the right, and *vice versa*, plane 3 corresponding to plane 3 on the other side).

The three semicircular canals on either side lie in planes that are roughly (not exactly) perpendicular to one another.

The horizontal semicircular canal is not quite horizontal. In the usual erect position of the head it slopes backwards at an angle of about 30 degrees.

FIG. 47.—DIAGRAM SHOWING THE DIFFERENT PLANES OF THE SEMICIRCULAR CANALS (SEEN FROM BEHIND).

1. Superior or anterior } both nearly vertical.
2. Inferior or posterior }
3. External or horizontal semicircular canals.

Any movement of the endolymph in relation to the semicircular canals or *vice versa* is called *lymphokinesis*, and constitutes the irritation which stimulates the semicircular canal or canals in which the lymphokinesis takes place. The direction of the lymphokinesis is always said to be the direction of current of the endolymph in relation to the semicircular canals, even if it really is a primary movement of the canals in relation to the stationary endolymph.

The Rotation Test.

When the patient is rotated with his head bent slightly forward (about 30 degrees), lymphokinesis takes place in the horizontal semicircular canals, these being moved in relation to the endolymph, which to start with hangs back. Gradually, however, the endolymph gathers momentum, and when the rotation after a little while is stopped, the endolymph, on account of its momentum, continues to move in the direction of the rotation. Thus, during rotation lymphokinesis takes place in the direction opposite to that of the rotation, whilst after the rotation lymphokinesis takes place in the same direction as the rotation. The phenomena due to the first phase of lymphokinesis escape observation. The phenomena we observe are all due to the second phase—*i.e.*, the lymphokinesis in the direction of the rotation.

This causes:

(1) Horizontal nystagmus in the opposite direction.
(2) Past-pointing
(3) Postural deviation (*i.e.*, tendency to fall)

} in the direction of rotation.

The horizontal semicircular canals are irritated on both sides, but the one opposite to the side towards which the rotation has taken place is being subjected to a more intense irritation than the other. Consequently, when the labyrinth on the one side is affected, rotation in the direction towards this side will give a more marked reaction than to the other side.

Any difference in the duration of the post-rotatory

nystagmus will therefore be of interest, and ought to be noted.

Normally the nystagmus lasts twenty to thirty seconds. After this time all the phenomena have disappeared, and if, after having timed the elicited nystagmus, it is necessary to observe also the other phenomena (past-pointing, tendency to fall), renewed rotation is required. These latter phenomena are, however, as a rule more conveniently studied in connection with

The Caloric Test.

When the meatus externus is syringed with cold water, or cold air blown into it, the temperature is lowered in the part of the semicircular canals which are most

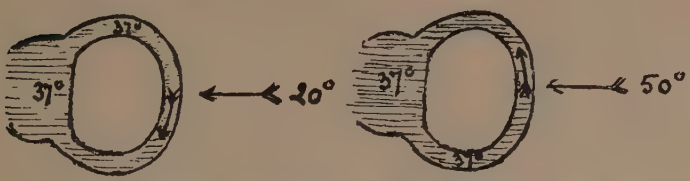

FIG. 48.—DIAGRAM TO ILLUSTRATE THE CALORIC TEST WITH COLD (20°) AND HOT (50°) WATER.

The arrow outside the semicircular canal indicates the thermic influence in either case. The arrow inside the semicircular canal indicates the direction of the resulting lymphokinesis. The temperature is given in centigrades.

laterally situated. The endolymph in this part will sink down, and thus cause lymphokinesis (according to the same thermosyphon principle which is utilized in water-cooling on light motor-cars). When hot water is used, lymphokinesis in the opposite direction is set up (*cf.* Fig. 48).

The optimal position for eliciting this caloric lymphokinesis in a semicircular canal is the vertical position. In order to place the horizontal canal in this position the patient ought to bend his head backwards about 60 degrees, which is the position of the head usually assumed when the patient lies in bed or on a couch with a small pillow under his head.

If we perform the caloric test in the upright position, the horizontal canal is far removed from the optimal position. We get a faint horizontal nystagmus, and mixed with this a stronger rotatory nystagmus as a result of stimulation of the other two semicircular canals, which are now practically vertical.

By taking the position of the semicircular canals into account (*cf.* Fig. 47) we can thus, by altering the position of the head by means of the caloric test, elicit a great number of different forms of nystagmus.

By syringing the right ear with cold water in the recumbent position we normally elicit:

(1) Nystagmus (horizontal) to the left.
(2) Past-pointing to the right.
(3) Slight tendency to fall to the right.

By doing the same in the sitting position:

(1) Nystagmus to the left, chiefly rotatory.
(2) Past-pointing to the right.
(3) Marked tendency to fall to the right, the postural deviations as elicited by the Romberg test being chiefly a result of lymphokinesis in the superior semicircular canals.

Total absence of vestibular reaction (no nystagmus, no past-pointing, no postural deviation in the Romberg position) is indicative of destruction of the labyrinth or the extra-bulbar part of the vestibular nerve.

Absence of nystagmus by otherwise normal vestibular reaction (past-pointing and postural deviation) points to a lesion of the fasciculus longitudinalis posterior.

Absence of past-pointing by otherwise normal reaction points to a cerebellar lesion. When a spontaneous past-pointing is found to disappear by vestibular irritation, this is, according to Barany, an indication that the original past-pointing was due to irritation of cerebellar directional centres.

V. ANATOMICAL DIAGRAMS.

The following diagrams are meant as anatomical "aids to memory" only, giving a rough orientation of the most essential anatomical facts:

FIG. 49.—DESCRIPTION OF DIAGRAM OF VOLUNTARY MOTOR SYSTEM.

In the precentral gyrus of the brain the various motor centres are situated. From above downwards they are found in the following order: foot, leg, thigh, trunk, shoulder, over-arm, forearm, hand, face, lips, and tongue.

From this gyrus the pyramidal fibres converge towards the internal capsule, and descend through this structure (anterior to the sensory paths) and through the anterior part of the brain stem. Here the fibres for the motor cranial nerves leave the main pyramidal tract and cross over to the motor nuclei on the other side. All the motor cranial nuclei except the seventh and the twelfth have a bilateral cortical innervation, however, and also the upper portion of the facial nucleus has a bilateral cortical innervation.

In the medulla oblongata most of the motor fibres cross to the lateral column on the other side (decussatio pyramidum).

Besides the motor paths represented here (the pyramidal tracts, which are the paths of *voluntary* innervation) there are also other motor paths of emotional and reflex innervation (rubro-spinal tracts and others).

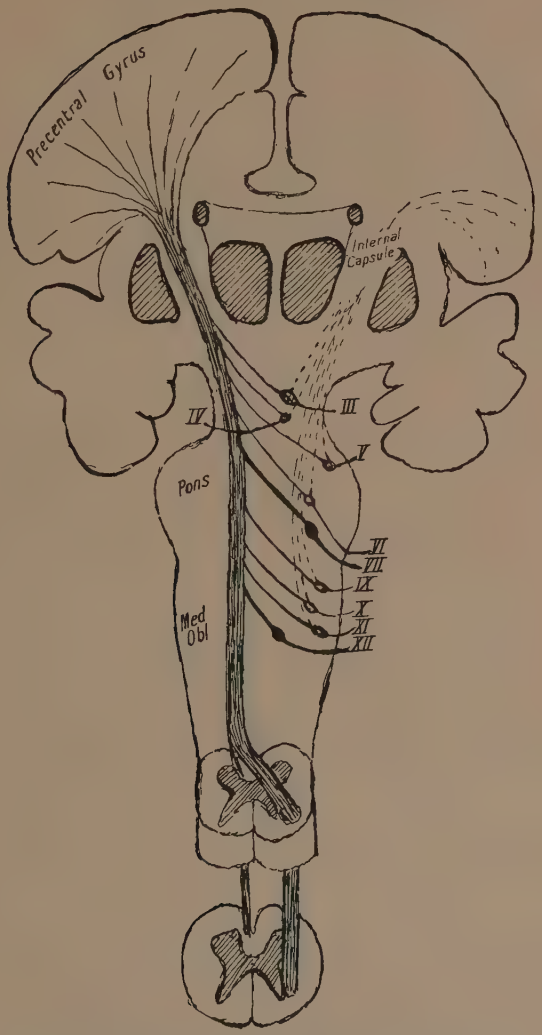

FIG. 49.—DIAGRAM OF VOLUNTARY MOTOR SYSTEM.

FIG. 50.—DESCRIPTION OF DIAGRAM OF SENSORY SYSTEM.

The fibres conducting sensation of superficial pain (pin-pricks) and temperature cross immediately or very soon* after they have entered the cord to the antero-lateral column. The fibres conducting the stimuli of deep sensation ascend in the posterior columns without crossing to the nuclei of the posterior columns in the medulla oblongata. Here they cross and ascend to the optic thalamus, joining the fibres conducting pain and temperature. The impressions of light touch are conveyed by both routes (partly crossing in the segment of entrance, to the anterior column of the other side, partly following the path of deep sensation; see also Fig. 51). In the distribution of the tactile conduction between these two routes there are individual variations: in some individuals the tactile impressions are conducted chiefly through the posterior columns (uncrossed up to medulla oblongata); in others chiefly through the anterior columns (crossed); in others, again, equally distributed between the two routes.

From the optic thalamus the sensory impressions are conducted mainly to the postcentral gyrus, where a localization roughly corresponding to the motor localization in the precentral gyrus is found: the centres for the opposite lower limb at the superior end of the gyrus, the centres for the opposite upper limb and the head at the inferior end of the gyrus. It is doubtful whether the sensation of pain has a cortical localization. Probably the final registration of painful stimuli takes place in the optic thalamus, which seems to be part of the anatomical substratum of the emotional functions. The cerebral cortex is mainly concerned with the more discriminating parts of sensation.

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* The fibres conducting sensations of temperature probably cross a few segments higher up than the fibres conducting pain. It is doubtful if any crossing takes place below the twelfth dorsal segment.

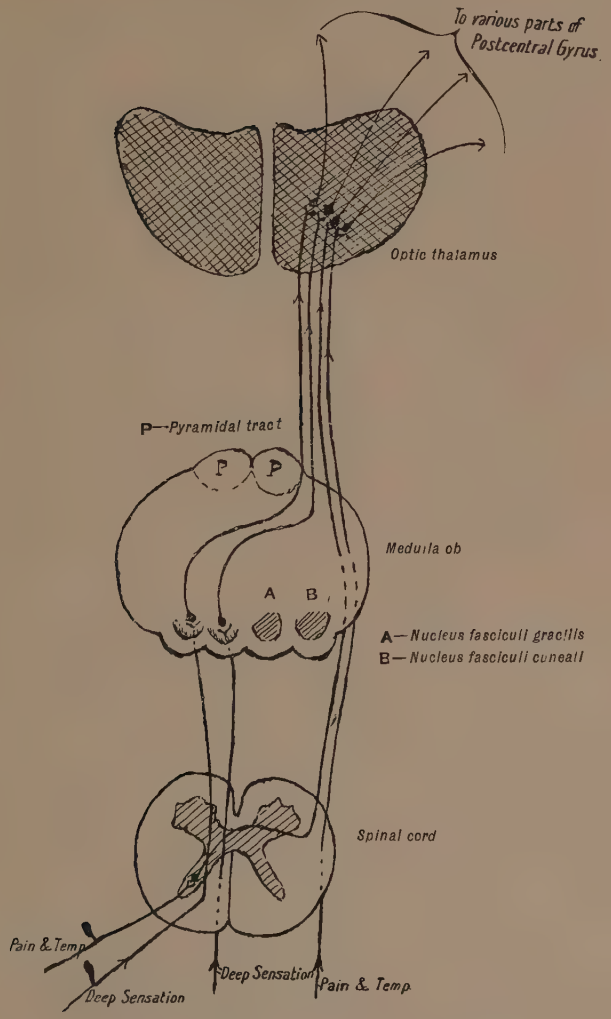

FIG. 50.—DIAGRAM OF SENSORY SYSTEM.

FIG. 51.—DESCRIPTION OF DIAGRAM OF SECTION OF SPINAL CORD.

1. Anterior grey horns (cells of the peripheral motor neurons).
2. Pyramidal tracts (tractus cortico-spinalis, voluntary movements).
   2, *a*, crossed.
   2, *b*, uncrossed (disappearing in cervico-dorsal cord).
3. Tractus spino-thalamicus.
   3, *a*, of the lateral column (temperature and pain).
   3, *b*, of the anterior column (tactile).
4. Tractus rubro-spinalis (? emotional).
5. Tractus spino-bulbaris (deep sensation and tactile).
   5, *a*, fasciculus gracilis (Goll).
   5, *b*, fasciculus cuneatus (Burdach).
6. Cerebellar tracts.
   6, *a*, direct.
   6, *b*, indirect.
7. Central canal.

ANATOMICAL "FORMULÆ" OF SOME DISEASES AFFECTING THE SPINAL CORD.

Progressive muscular atrophy and poliomyelitis ant. acuta; 1.
Amyotrophic lateral sclerosis: 1+2.
Tabes: posterior roots+5.
Subacute combined sclerosis: 2+5
Friedreich's disease: 2+5+6.

The above "formulæ" refer to so-called "*system lesions*" of the cord. For the chief types of "*non-system lesions*" see p. 186.

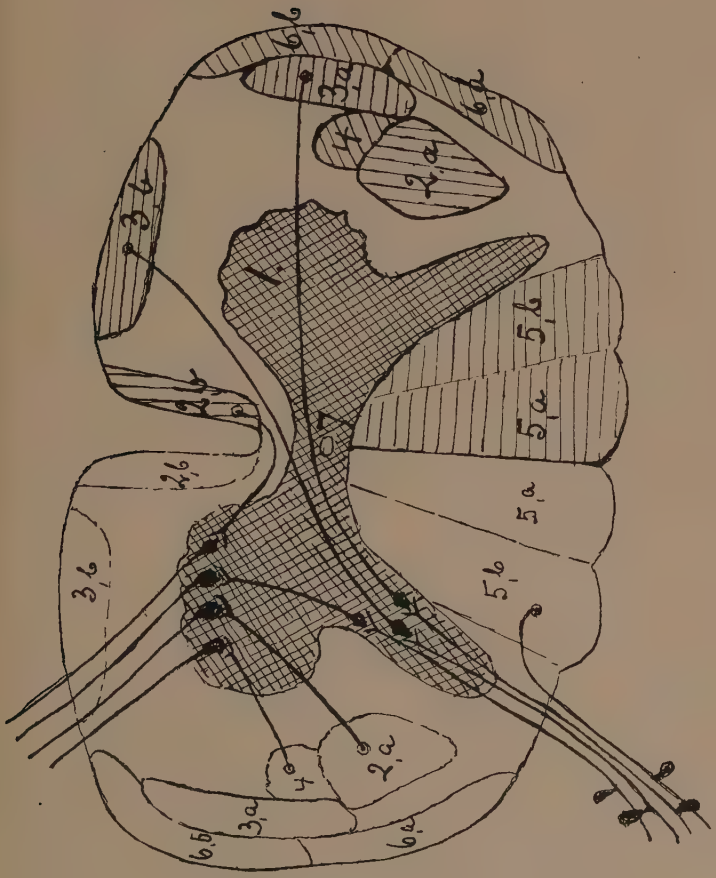

FIG. 51.—DIAGRAM OF SECTION OF SPINAL CORD.

CHIEF TYPES OF "NON-SYSTEM LESIONS" OF THE SPINAL CORD

I. *Transverse Lesion*.—According to the intensity of the lesion* the clinical picture varies a great deal owing to the difference in "functional vulnerability"† of the different tracts. These give up their function in the following order:

1. The pyramidal tracts (Fig. 51, 2 *a*, *b*) (the youngest both in phylogenetic and ontogenetic respect) are the first to suffer. This results in bilateral paresis of pyramidal type of all segments of the body, caudal to the lesion (spastic paraplegia).

2. The posterior columns (Fig. 51, 5 *a*, *b*) are the next to suffer. Result: Ataxia and diminution of deep sensation.

3. The spino-thalamic tracts (Fig. 51, 3 *a*, *b*) then give in. Result: Loss of superficial sensation from all segments, caudal to the lesion.

II. *Central Lesion* (*i.e.*, lesion confined to central grey matter —*e.g.*, intramedullary tumours, early cases of syringomyelia):

(*a*) Atrophic, flaccid paralysis (Fig. 51, 1) } in corresponding segments only.
(*b*) Loss of superficial sensation (all the crossing fibres in Fig. 51) }

III. *Brown-Séquard Lesion* (*i.e.*, lesion limited to one half of the cord:

(*a*) Homolateral paresis of pyramidal type } in all segments caudal to the lesion.
(*b*) Homolateral loss of deep sensation }
(*c*) Heterolateral loss of superficial sensation (pain and temperature) }
(*d*) Homolateral root phenomena (*i.e.*, atrophic paralysis and loss of all qualities of sensation in corresponding segments only), often missing.

Pure Brown-Séquard syndromes are rare. Transverse lesions may often show some elements of a Brown-Séquard lesion, the motor loss being more pronounced on the one, the superficial sensory loss more pronounced on the other side, thus indicating that the one half of the cord (that of the chief motor loss) is more severely affected than the other (*e.g.*, uneven pressure upon the cord from an extramedullary tumour).

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* A complete transverse lesion of the cord is a rare condition.

† This is not necessarily identical with anatomical vulnerability.

CHIEF TYPES OF HEMIPLEGIA

When a Brown-Séquard lesion (see p. 186) is situated above the cervical intumescence, (A) a *spinal hemiplegia* ensues. The two other chief types of hemiplegia are:

(B) The *crossed hemiplegia* (hemiplegia alternans), caused by a lesion in the brain stem;* this syndrome consists of (*a*) cranial nerve disturbance on one side (the side of the lesion) and (*b*) hemiplegia affecting the limbs of the other side. According to the level of the lesion various cranial nerves are affected and correspondingly various types of crossed hemiplegia are distinguished (Weber's syndrome, Millard-Gubler's syndrome, Jackson's syndrome, and others).

(C) The *ordinary hemiplegia*, caused by a lesion in the capsula interna. This hemiplegia is sometimes accompanied by facial and hypoglossal paresis of central type. All the motor (and possibly accompanying sensory)† loss is confined to one side, the one opposite the lesion (*cf.* Fig. 49, p. 181).

In right hemiplegia aphasia of predominantly motor type is a fairly common complication due to an extension of the lesion to the speech area in the left hemisphere.

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* *I.e.*, a unilateral lesion of the *ventral* (anterior) part of the brain stem. The sensory paths are to be found behind the pyramidal tracts. Lesions more dorsally situated, therefore, may produce corresponding sensory syndromes (crossed hemianæsthesia).

† The sensory paths are to be found in the posterior parts of the internal capsule in close relation to (partly intermingled with) the pyramidal fibres for the leg.

FIG. 52.—DESCRIPTION OF DIAGRAM OF CORTICAL LOCALIZATION.

(The indications in the frontal part of this diagram should be regarded with a certain reservation, as our views regarding these are still under discussion.)

The arrangement of the different motor centres in the precentral gyrus gives the key to the understanding of "cortical epilepsy," or "Jacksonian attacks," as these always progress in the order of cortical topography—*i.e.*, the convulsions spread from area to area exactly corresponding to the arrangement of the different motor centres in the precentral gyrus.

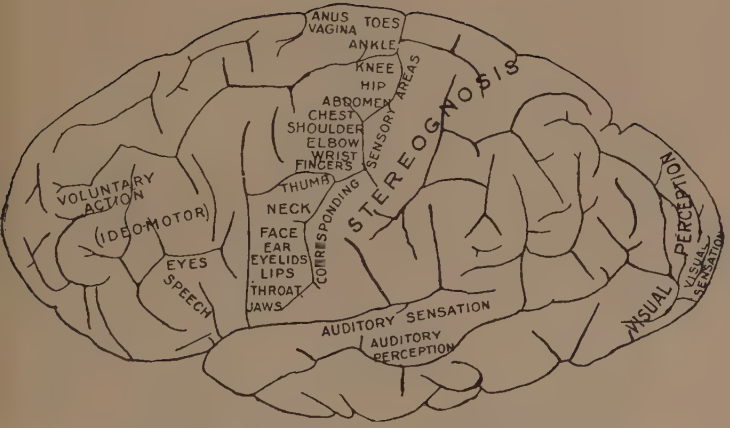

FIG. 52.—DIAGRAM OF CORTICAL LOCALIZATION.

FIG. 53.—DESCRIPTION OF DIAGRAM OF THE VEGETATIVE NERVOUS SYSTEM.

The dorso-lumbar sympathetic system contains within the intermedio-lateral columns of the spinal cord vasomotor (and pilomotor) centres for the whole body. The segmental arrangement of these centres is *quite different from the sensori-motor segmentation.* The head and the neck are innervated from D_1-D_4, the upper limbs from D_4-D_9 (or D_{10}), and the lower limbs from D_{11}-L_2. A lesion in the mid-dorsal region of the cord may therefore cause vasomotor disturbances in the upper limbs; these vasomotor disturbances may be accompanied by paresthesiæ (feeling of numbness) and even of slight diminution of tactile sensation, thus simulating a lesion of the lower cervical region of the cord (Barré, Schrapf, Guillain).

DIAGRAM OF THE VEGETATIVE NERVOUS SYSTEM

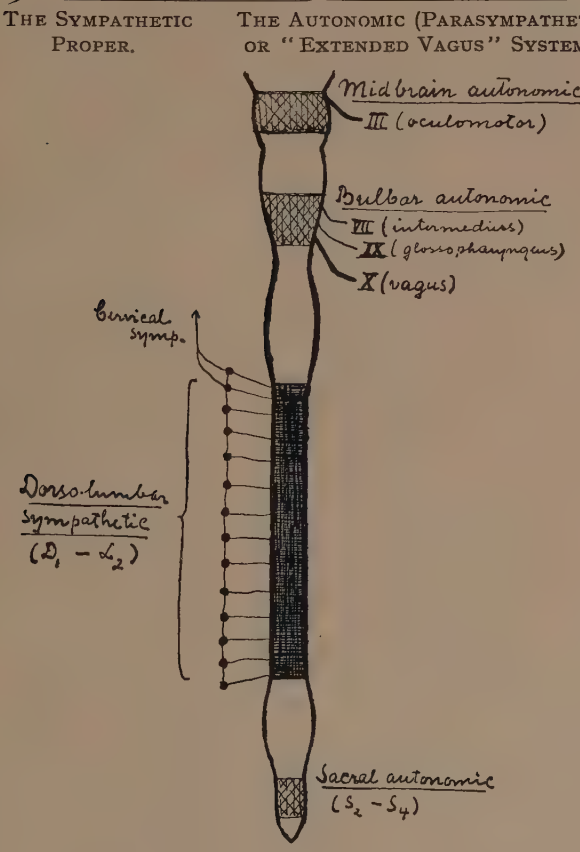

FIG. 53.

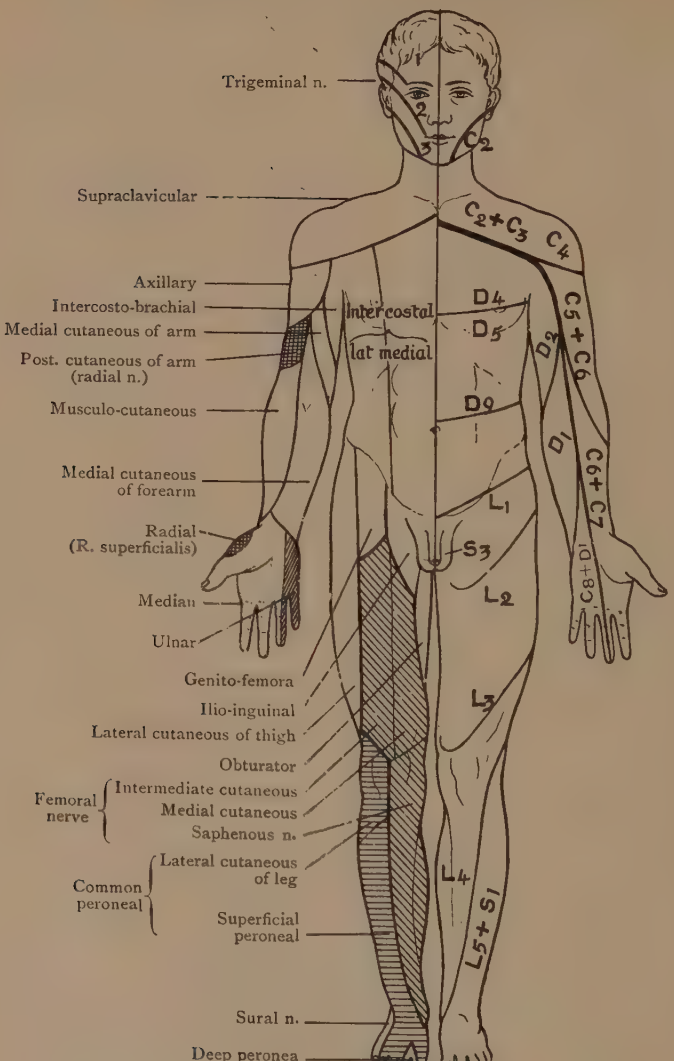

FIG. 54.

DIAGRAMS OF SENSORY SEGMENTAL (LEFT HALF OF BODY) AND PERIPHERAL
(Considerable individual

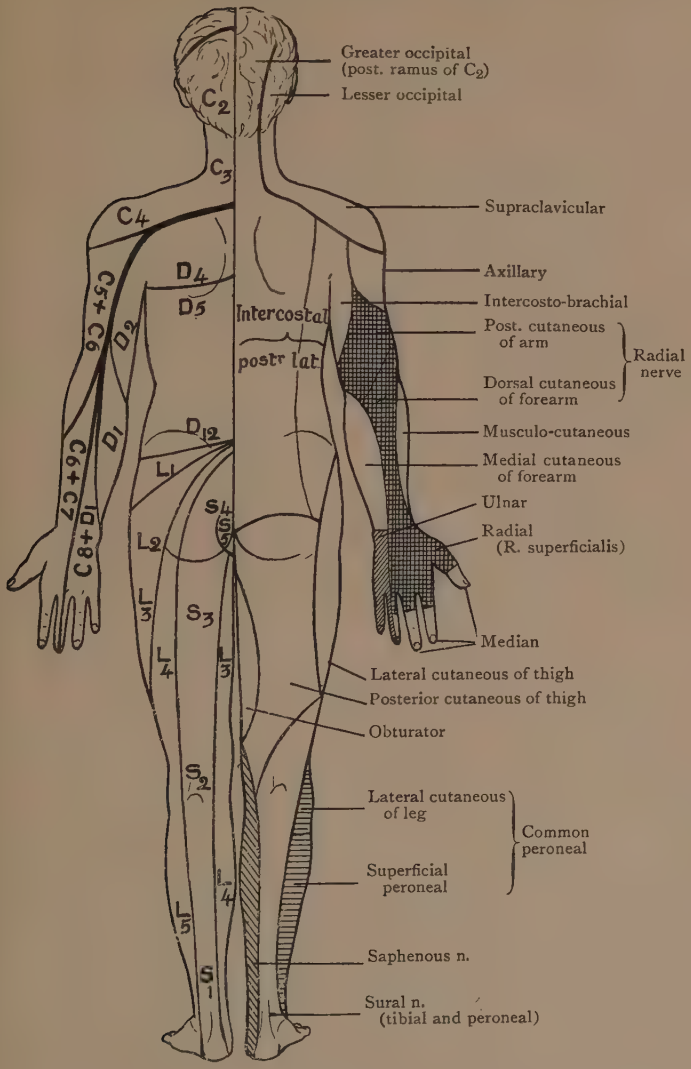

FIG 55.

NERVE DISTRIBUTION (RIGHT HALF) OF THE SURFACE OF THE BODY. variations in respect of both.)

VI. PHARMACOLOGICAL TESTS OF THE VEGETATIVE NERVOUS SYSTEM.

The vegetative nervous system may be subdivided into two different systems: (1) the sympathetic proper or thoracico-lumbar system; and (2) the parasympathetic or autonomic or "extended vagus" system (*cf.* diagram, Fig. 53, p. 191).*

Whilst the thoracico-lumbar or sympathetic system supplies nerve fibres to all parts of the body, the parasympathetic supplies nerve fibres to special regions only. Of the three divisions of the parasympathetic system (i.) the mid-brain (tectal) autonomic system supplies the sphincter iridis and the ciliary muscle; (ii.) the bulbar (vagus) autonomic system supplies the heart, the lungs, the lachrymal and salivary glands, the œsophagus, stomach, liver, pancreas, the small intestine, and the anterior part of the large intestine; (iii.) the sacral autonomic system innervates the lower part of the large intestine, the bladder, and the external sexual organs (penis and vagina).

All these parts just mentioned thus receive a double vegetative nerve supply, being supplied both from the sympathetic and the parasympathetic systems. Experi-

* To these two divisions may be added a third: (3) the intramural vegetative nervous system, consisting of nerve cells and fibres enclosed in the peripheral organs (*e.g.*, the heart, the intestines). It seems capable of a certain automatism. Its relation to the other two divisions (probably one of subordination) is not quite clear.

ments have shown that this double innervation is antagonistic in nearly all respects—*i.e.*, where irritation of the sympathetic (thoraco-lumbar) fibres have a certain effect, irritation of the parasympathetic fibres have the opposite effect.*

The " tonus " and the irritability of these two systems vary greatly from individual to individual. In some individuals and in some pathologic conditions the tonus of the sympathetic (thoracico-lumbar) system is preponderating, and we speak of *sympaticotonic conditions* (*e.g.*, hyperthyroidism, Graves' disease); the irritability of the sympathetic is then increased correspondingly.

In other individuals and in other pathologic conditions the " tonus " and the irritability of the parasympathetic (extended vagus) system are increased, and we then speak of *vagotonic conditions* (*e.g.*, bronchial asthma, spastic constipation, mucous colitis, possibly some cases of gastric hypersecretion and gastric ulcer).†

It is therefore of great clinical interest to determine the relative irritability of these two vegetative systems. This can be done by means of certain drugs, which are known to have an irritating effect on either of the

* This antagonism seems to be rather complicated, however, inasmuch as irritation of one system is often (? always) accompanied by increased irritability of the other. Thus, when the sympathetic has been stimulated by means of adrenaline, the action of calcium on the parasympathetic seems to be increased (Billigheimer).

† Pure, clean-cut, and complete cases of sympathicotonia and vagotonia are rarely (if ever) to be seen in the clinic. It is questionable, therefore, if the two terms, " vagotonia " and " sympathicotonia " (Eppinger and Hess), should not be replaced by the less committing term " vegetative stigmata " (G. v. Bergmann).

two systems, and thus elicit reactions, which can easily be controlled.

Of a number of drugs, all influencing the vegetative nervous system, the two chief ones are adrenaline, which stimulates the nerve endings of the sympathetic (thoracico-lumbar) system, and pilocarpine, which stimulates the nerve endings of the parasympathetic (extended vagus) system. Muscarine and physostigmine have a similar action to pilocarpine. Ergotoxine paralyzes the nerve endings of the sympathetic, atropine those of the parasympathetic. Nicotine has a paralyzing action on the synapses between the preganglionic and postganglionic fibres of both systems.

For clinical tests we use (i.) adrenaline, (ii.) pilocarpine, and (iii.) atropine only.

(i.) **Adrenaline** may be used for conjunctival application (Löwi's reaction). Two or three drops of 1 in 1,000 adrenaline solution are placed in the conjunctival sac. Only in sympathicotonic conditions (hyperthyroidism, after hypodermic cocaine injection) a dilatation of the pupils occurs. (This mydriasis is also seen in certain cases of diabetes [?] of pancreatic origin.)

Obviously the size of the pupils should be examined under exactly the same conditions of light and accommodation before and after the adrenaline application. This may be difficult, in daylight sometimes impossible. The test applied to both eyes simultaneously is thus not quite reliable. It is therefore better to *apply the adrenaline solution to one eye only, utilizing the pupil of the other eye for comparison.* After the adrenaline instillation the pupils should be inspected every five or ten minutes

for several hours;* and then, in case of mydriasis occurring, they should be inspected every two hours during the whole day.

Adrenaline is also used hypodermically (1 milligramme) and intravenously (0·01-0·03 milligramme).

The results of such injections are normally:

(1) Vasoconstriction† betraying itself by pallor of the face.

(2) Tremor, restlessness, feeling of anxiety and palpitations.

(3) Acceleration of the pulse (by about thirty beats a minute).

(4) Rise of blood-pressure (immediately after intra venous, within two to ten minutes after hypodermic injection).

(5) Increased frequency (sometimes irregularity) of respiration.

(6) Increase in the amount of blood-sugar (maximum ten minutes after intravenous, thirty minutes after hypodermic, injection).

(7) Glycosuria only in sympathicotonic conditions (examination of urine every two hours for six hours).

(8) Increased number of lymphocytes in the blood.

(9) Increased irritability of the pilomotor reflexes or pilomotor contraction. (Lewandowsky has found that adrenaline causes erection of the quills of the hedgehog.)

In sympathicotonic conditions these symptoms are

* According to Söderbergh, the reaction in some cases does not appear till after five hours (!). (*Réunion Neurologique*, 1926.)

† The cerebral and coronary arteries do not participate in this vasoconstriction. On the contrary, they seem to dilate (Langendorff).

increased; in vagotonic conditions decreased; in some instances reversed in regard to the pulse-rate.

(ii.) **Pilocarpine** is also injected hypodermically (1 centigramme) and intravenously (7·5 milligrammes).

In normal individuals the effects of such injections are:

Vasodilatation, betraying itself by redness of the face and feeling of heat.

Perspiration.

Increased secretion of saliva. (An amount of saliva exceeding 75 c.c. secreted during the first hour is considered as a vagotonic symptom.)

Increased secretion of tears and nasal mucus in vagotonic conditions only.

Pilocarpine also increases the formation of cerebro-spinal fluid. *Increased intracranial tension* should therefore be regarded as *contra-indication* to pilocarpine injections.

Where a transverse lesion of the cord has caused retention of urine and fæces, pilocarpine injections may procure evacuations of bladder and rectum.

Theoretically one would expect bradycardia, but as a rule the pulse is, on the contrary, somewhat accelerated, chiefly in vagotonic conditions.

Pilocarpine acts in all probability peripherally on the nerve endings of the parasympathetic (extended vagus) system, just as adrenaline acts on the sympathetic.

(iii.) **Atropine** injections, hypodermic (0·5 to 1 milligramme) and intravenous (0·5 to 0·75 milligramme) cause:

Acceleration of the pulse (twenty to thirty beats a minute).

Diminished secretion of saliva.

Diminished perspiration.

If the reaction to atropine injections is marked, and if at the same time such symptoms as bradycardia, respiratory arhythmia, or constipation disappear, these symptoms are to be regarded as vagotonic—*i.e.*, due to an overwhelming tonus of the parasympathetic (extended vagus) system.

Platz recommends these different tests to be carried out on three subsequent days: first day, intravenous injection of 0·01 milligramme* adrenaline, preceded and followed by pulse and blood-pressure readings every three minutes till return to normal, blood and urine tests for sugar; second day, intravenous injection of 0·75 milligramme atropine, followed by pulse readings; third day, intravenous injection of 7·5 milligrammes pilocarpine, followed by pulse readings and measuring of saliva during first hour. Palpitation, dryness of mouth and throat, and perspiration should also be recorded.

As the stimulating, respectively the paralyzing, effects of all these drugs take place peripherally, they cannot give us much information of the paths of conduction in the vegetative nervous system. This may, however, be obtained by other means, of which the examination of the pilomotor reflexes is the most important.

It will be seen that a drug stimulating the sympathetic and a drug paralyzing the parasympathetic may have the same effect. Therapeutically, therefore, adrenaline and atropine are expected to have the same beneficial effect in bronchial asthma: the first one

* Observations by Dr. Hval in my clinic show that adrenaline in this minute dosage cannot always be relied upon under normal conditions to produce a reaction. We therefore now use 0·02 milligramme for intravenous administration. Even this gives a faint reaction which is, however, fairly constant in normal individuals.

causing bronchodilatation by sympathetic stimulation, the latter causing bronchodilatation by parasympathetic (vagal) paralysis.

The antagonism between the sympathetic and the parasympathetic systems is perhaps not complete, nor is there an *absolutely* identical effect from adrenaline injection and experimental irritation of the sympathetic, from pilocarpine injection and irritation of the parasympathetic, etc., yet the parallelism is complete enough to be of some practical value. Also a number of other drugs have an influence on the vegetative nervous system (*e.g.*, cocaine).* It has also to be kept in mind that small amounts of the different drugs may cause effects different from those caused by larger amounts.

The following highly schematized *Table of the Pharmacologic Reactions of the Vegetative Nervous System* should therefore not be strained in its practical application.

TABLE OF THE PHARMACOLOGIC REACTIONS OF THE VEGETATIVE NERVOUS SYSTEM. (Chiefly after L. R. Müller.)

SYMPATHETIC SYSTEM (thoracico-lumbar)	PARASYMPATHETIC SYSTEM ("extended vagus")
IRRITATION (adrenaline):	IRRITATION (muscarine, pilocarpine, physostigmine):
Mydriasis	Miosis
Vasoconstriction	Bradycardia (missing in the pilocarpine test)
Pilomotor contraction	Increased salivation
Tachycardia	Increased perspiration
Bronchodilatation	Contractions of the intestinal canal
Inhibition of intestinal contractions	
PARALYSIS (ergotoxine):	PARALYSIS (atropine, scopolamin):
Vasodilatation	Mydriasis
Bradycardia	Tachycardia
	Bronchodilatation
	Decreased salivation
	Decreased perspiration
	Inhibition of intestinal contractions

* Cocaine irritates the sympathetic innervating the dilator pupillæ. Morphium irritates some of the centres of the "extended vagus" system (miosis, bradycardia). So does digitalis and calcium.

VII. ON THE INTERPRETATION OF X-RAY PICTURES OF THE SKULL

The cranium should always be X-ray photographed in two planes at least.

Alterations in the cranial bones themselves should first be looked for. Remember that the lamina interna of the cranial bones is a site of predilection for syphilitic affections,* sometimes showing in the radiogram. Remember, also, that bony changes (thickening as well as absorption) are often found over meningeal endotheliomata.

Increased intracranial tension sometimes betrays itself by "*convolutional thinning*" of the skull, produced by the pressure of the summits of the gyri. In the X-ray picture this may be seen as scattered areas of thinning, corresponding to the gyri ("finger-printing" or "beaten silver" appearance. This "beaten silver" appearance is normal in children and may remain till the twentieth or even twenty-fifth year according to Sosman). *Diastasis of cranial sutures* is a frequent finding in infants with increased intracranial tension (intracranial tumour, hydrocephalus).

General dilatation of diploic vein channels is regarded as a sign of increased intracranial pressure, but has in my opinion a doubtful value unless very pronounced.

A striking *local vascularity* is sometimes found in the

* Besides the tibia, the lamina interna of the flat cranial bones deserves the name of "os cheri de la syphilis" (Leri). *Cf.* Leri and Cottenot, *La Presse Médicale*, 1926, No. 51, p. 801.

cranial bone overlying a meningioma, and this increased local vascularity sometimes betrays itself in the X-ray picture. The presence of numerous and large meningeal channels in a circumscribed portion of the skull is therefore of diagnostic importance and points to the presence of a meningioma in this region. (It is also of prognostic importance, pointing to the danger of profuse bleeding during reflection of the osteoplastic flap.)

An *enlargement of the sella turcica* may be due to a pituitary tumour. This is as a rule the case, when there is a definite "ballooning" of the sella, or when there is destruction of the floor of the sella. But simple widening of the sella, without the distinctive features just mentioned, may only be the result of increased intracranial tension, caused by an intracranial tumour elsewhere (Souques and others). A tumour in the posterior cranial fossa frequently causes atrophy of the posterior clinoid processes.

The *dilatation of the porus acusticus internus* sometimes caused by acoustic tumours (Folke Henschen) is mostly difficult to ascertain in the ordinary radiograms "Diagonal" radiograms (patient's head resting with forehead, nose, and chin on the plate) from both sides may allow an approximate comparative judgment of the width of the porus on either side. A comparatively clear picture of the pars petrosa on either side may sometimes be had in a frontal picture, taken with the head in such a position as to project either pars petrosa into the orbita of the same side.* The internal auditory

* Stereoscopic X-ray pictures may also be of considerable help here.

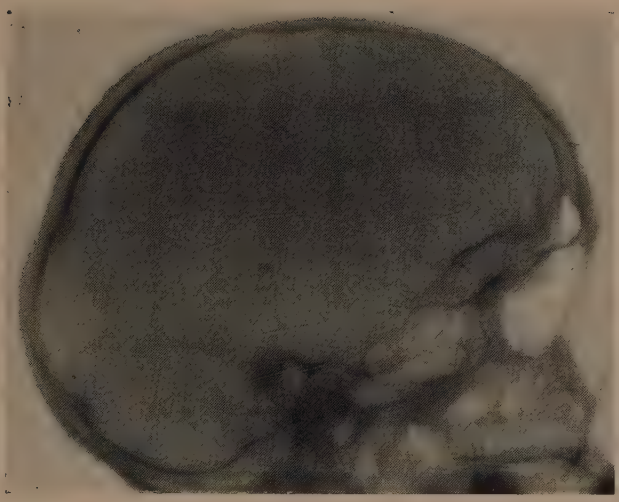

FIG. 56.—PINEAL SHADOW.

Sœthre and Jörgensen's publication of cases from my clinic (*Norsk Magazin for Lægevidenskaben*, 1926, No. 6, p. 425).

To face page 202.

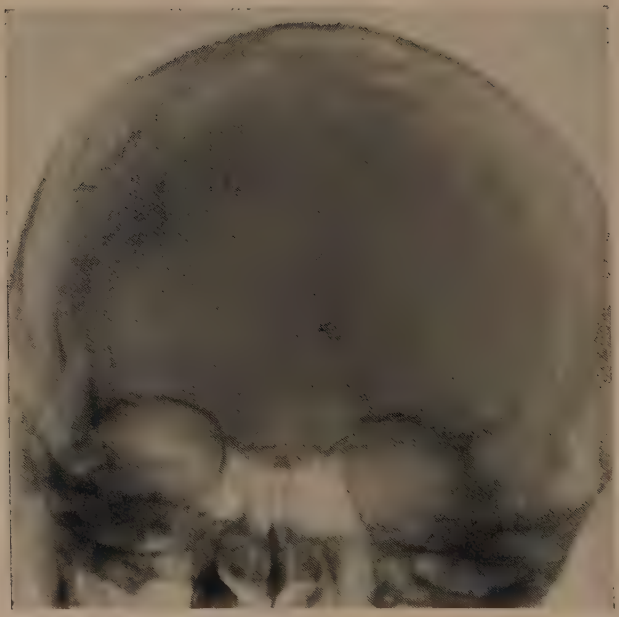

FIG. 57.—PINEAL SHIFT.

Dislocation of the pineal shadow to the left caused by tumour in the right hemisphere (from the same publication as Fig. 56).

To follow Fig. 56, *page* 203.

meatus then appears as a channel in the pars petrosa—not as a round hole.

The radiogram may also disclose *calcifications in the brain*. These may be indicative of tumours (*e.g.*, meningiomata of the olfactory groove, suprasellar meningiomata, cranio-pharyngeal poach cysts and others) or tuberculomata. They are, however, a quasi-normal finding in the pineal gland:* *A pineal shadow* is discovered in quite a considerable number of radiograms of adult skulls, and should always be looked for. A dislocation of the pineal shadow ("pineal shift") to one side is indicative of a tumour of fair size in the opposite hemisphere (*cf.* Figs. 56 and 57). Occasionally calcareous deposits are also seen in the falx cerebri.

Ventriculography and Encephalography.

In addition to the ordinary X-ray examination of the skull, one of the following two special methods may advantageously be employed in selected cases.

I. **Ventriculography.**—Air is introduced directly into one of the lateral ventricles of the brain, whereupon a series of X-ray pictures of the skull are taken.

It is a method which is not without its danger (from some clinics quite an appreciable mortality is reported), and it should therefore not be used as a routine method, and should only be carried out by someone who has

* Apparently even an extensive calcification of the pineal gland is followed by no clinical disturbances—at least not in adults. Only in young boys does a lesion of the pineal gland lead to manifest clinical disturbances ("pubertas præcox" or "macro-genitosomia præcox"; *cf.* p. 17, footnote).

sufficient experience in cerebral surgery to cope with the dangers that may arise.

Experience has led me to take a rather unorthodox position in regard to ventriculography. In many clinics it is the aim to get both ventricles filled as much as possible with air. As far as I can see, the first consideration should be *not to inflate too much air*.

Even with only 20 to 25 c.c. of air inflated a fairly complete impression of the state of both ventricles can be obtained; only it requires a larger series of X-ray pictures in different positions. Bearing in mind that the air will always seek the highest points of the ventricular system, it is easy even with a very limited amount of air inflated to ascertain (1) the patency or obstruction of the foramen of Monro and (2) to study the complete outline of both lateral ventricles.

The minimum of X-ray pictures needed will be seven—viz.: (1) First one with the rays in sagittal direction when the patient has been lying for ten minutes on the side, into the lateral ventricle of which the air has been inflated. This will show whether the foramen of Monro is patent or not, and also if the lateral ventricles are displaced. (2) Then three pictures from either side with different inclination of the head so as to expose the different portions of either lateral ventricle (anterior, middle, and posterior portion).

In different clinics different routes are chosen for punctures of the lateral ventricle.

One of the two following methods is preferable:

(i.) Puncture of the posterior horn of the lateral ventricle. A small trephine opening is made 2 to 3 cm.

lateral and superior to the external occipital protuberance. The puncture needle is then introduced in the direction of the centre of the homolateral eye of the patient. At a depth of about 4 cm. the posterior horn is normally encountered. (ii.) Kocher's method. A trephine opening is made just in front of the bregma, 2 to 3 cm. from the mid-line, so as to avoid the sagittal sinus. A line drawn upwards from the external auditory meatus at right angles with a line from the meatus passing just below the nose will approximately indicate the position of the bregma. The puncture needle should be introduced parallel to the median plane and somewhat backwards (roughly parallel to the line just indicated from the external auditory meatus to the bregma). At a depth of about 5 cm. the lateral ventricle is encountered. Both methods can conveniently be carried out bilaterally.

The trephine opening should be large enough to give a fair view of a small area of the dura in order to enable the operator to avoid dural and cortical vessels in choosing the point of penetration.

Local anæsthesia (novocaine 1 per cent.) of skin and pericranial structures suffices to make the whole procedure painless.

When the dura has been pierced the stylet is removed and the brain tissue is punctured with the hollow needle, which, as Penfield and Buckley* have shown, causes a much smaller scar in the brain than puncturing with a blunt needle.

* Proceedings of fifty-third meeting of the American Neurologist Association, Atlantic City, May 24, 1927

50 c.c. or more of cb.-fl. should be removed, preferably not by aspiration (the table should be tilted so as to place the puncture opening at a lower level than the spinal subarachnoid space), and 20 to 30 c.c. of air then inflated slowly and gently. After the inflation the puncture needle should be left in place for a little while for any increased pressure to be let off. (*Cf.* the expansion of the injected air when heated to body temperature.)

The advantage of ventriculography is a clear picture of the ventricles—of each ventricle separately, when a small amount of air has been inflated, which is also to be recommended on the grounds of causing less danger to the patient and thus lowering the mortality (which has been calculated by Grant to be 8 per cent.—somewhat high for a purely diagnostic proceeding !).

The ventriculography, however, gives no information as to the condition of the subarachnoid space over the hemispheres. In order to obtain this, encephalography should be carried out.

II. **Encephalography.**—This method, which has been used both for diagnostic and therapeutic* purposes, has the disadvantage of causing the patient much more discomfort than ventriculography. It has generally been considered much less dangerous. Bingel in 1922 reported 250 cases of encephalography without a fatal accident; but already the following year the same author

* *Cf.* Penfield: "Chronic Meningeal (Post-Traumatic) Headache and its Specific Treatment by Lumbar Air Insufflation: Encephalography," *Surgery, Gynecology and Obstetrics*, December, 1927, pp. 747-759.

reports no less than fifteen deaths due to encephalography.*

It must be considered to be absolutely contra-indicated in tumours in the posterior cranial fossa.

In this method air is injected by the lumbar route. 5 c.c. cb.-fl. is removed and replaced by 3 to 4 c.c. air; then a further 5 c.c. of cb.-fl. is removed and replaced by 3 to 4 c.c. of air; again 5 c.c. fluid withdrawn and replaced by air—and so on until 100 to 120 c.c. of cb.-fl. have been removed and replaced by 75 to 85 c.c. of air.

In encephalography the air enters the subarachnoid space on the hemisphere and also (to a varying extent) into the lateral ventricle, when the foramina of Magendie, the foramina of Luschka, the Sylvian aqueduct and foramen of Monro are patent.

The patient should be lying on his side on a sloping table (head at the top). By means of various positions of the head one can to a certain extent direct the air chiefly to the lateral ventricles or chiefly into the various areas of the subarachnoid space of the convexity.

Ascending lipiodol has also been utilized for encephalography, but it cannot compete with air injections in this respect.† Also it may possibly cause obstruction of the villi chorii.

* *Cf.* Bingel: "Todesfälle nach Gaseinblasung in den Lumbalkanal bzw. in die Gehirnventricel," *Med. Klinik*, 1923, Bd. 19, S. 637.

† The method of injecting Iodin Solution into the internal carotid (Egaz Moniz) in connection with X-ray photography can only just be mentioned here. I have as yet no personal experience of this method.

VIII. ON REPEATED EXAMINATIONS.

As in the neurological clinics the patients are examined over and over again by the same or different observers, several elements are introduced preventing the results of the different examinations from being completely comparable, even when carried out by the same observer.

It has already been mentioned that fatigue and a flagging attention may influence the exactness and the result of the examination.

In repeated examinations another factor must be taken into account, viz.: *practice*.

All motor and sensory functions can be perfected by practice, the more so the more complicated they are. This applies whether the functions be normal or pathologically disturbed. The practice need not be very extensive in order to influence the function—*e.g.*, everyone can find out for himself how an hour's practice with the compass points refines his sense of discrimination (*cf.* p. 81). As shown by Volkmann and Fechner, this practice, when carried out in a limited field of the body surface, improves the sense of discrimination not only in this field, but also in the adjacent area and the corresponding region of the other side of the body.*

Obviously, then, repeated examinations constitute in themselves a kind of unintentional re-education. This is most pronounced in examinations of the psychosomatic functions, the discriminating and complicated

* *Cf.* William James, "Principles of Psychology," vol. i., p. 514.

forms of sensation and co-ordination. As regards the latter, it has to be kept in mind, as already hinted at (p. 74), that in co-ordination disturbances of cerebellar origin, the patients very soon attempt a kind of correction often resulting in over-correction, which in the deviation tests (*cf.* pp. 75-76), may be bewildering.

From what has been said, it will be easily seen that improved functional findings at a later examination (even when carried out by the same observer) do not necessarily indicate an exactly corresponding anatomical improvement. Allowances must always be made for the element of unintentional re-education.

Some patients with increased intracranial pressure (either on account of intracranial tumour or of cerebral œdema in consequence of head injury) may on admission be unconscious or so dazed that the first examination will be incomplete.

An attempt should then be made to lower the intracranial pressure so as to make the patient more co-operative for a subsequent examination (whereby also important therapeutic indications are fulfilled).

This can be done by administration of hypertonic solutions—viz.:

1. By *intravenous* injection of—
   - (α) 60 to 100 c.c. of 15 per cent. to 20 per cent. solution of sodium chloride (maximal dose: 100 c.c. of 30 per cent. solution), or
   - (β) 50 per cent. solution of chemically pure glucose in normal saline solution.

The intravenous injection must in either case be carried out very slowly (at the rate of 2 to 3 c.c. a minute; thus an injection of 60 c.c. should take 20 minutes or more).

2. By *enemata* of 200 c.c. of 40 per cent. solution of magnesium sulphate, or, if this strong solution is found too irritating, 300 c.c. of 30 per cent. solution. The enema must be retained for at least half an hour.

3. By *oral administration of magnesium sulphate*: 100 c.c. of a 50 per cent. solution (often very nauseating), or repeated doses of 2 to 5 grammes of dry magnesium sulphate. (A mild, more protracted effect than 1 and 2.)

The patient's intake of fluid should be restricted, in order to obtain the full effect of any of the above methods.

IX. ON THE FIRST ROUTINE EXAMINATION

It is obvious that all the different tests described in the preceding pages cannot be brought into the first routine examination of patients on their admission to hospital, at their first visit in the out-patients' department or in the private consulting room. The beginner will sometimes experience difficulty in deciding what to comprise in this first examination.

Hard-and-fast rules cannot be given; the examination must, of course, be varied to suit the case; but as a rough guide for the routine examination of a patient on admission to hospital the following list may serve:

(1) Mental state: rough impression of patient's attention, orientation, perception, memory, reasoning powers, emotional control, and psychomotor reactions, obtained by simply conversing with the patient during the examination (the special tests for attention, etc., being reserved for subsequent examinations).

(2) Cranial nerves, with the exception of the vestibular tests. Where there is no facial paralysis the examination of taste need not be included in the first examination. *N.B.*—Articulation (*cf.* pp. 14-51).

(3) Motor system and co-ordination, special tests for cerebellar signs and study of combined movements being postponed for subsequent examinations (*cf.* pp. 53-66 and 71-80).

(4) Sensory system: rough orientation as to the sense of touch, temperature, and pain (superficial and deep pressure pain); joint sense (finger-heel-pointing test). Stereognostic sense. Do not insist upon details during the first examination if the patient is at all tired (*cf.* pp. 79-82, 83-85 and 87).

(5) Reflexes (*cf.* pp. 92-106 and 111):

Jaw-jerk.
Radialis periosteal reflex (radial and ulnar pronator jerk).
Biceps-jerk.
Triceps-jerk.
Patellar-jerk.
Ankle-jerk.
Abdominal reflexes.
Cremasteric reflexes.
Plantar reflexes.
Pathological shortening reflex.

A closer study of reflexes of spinal automatism, postural reflexes and organic reflexes is postponed for subsequent examinations.

(6) Standing position (Romberg's sign) and gait (*cf.* pp. 118 and 119).

This first routine examination will then, of course, always have to be completed in different ways according to the nature of the case.

For the first examination and note-taking in the out-patients' department and in the consulting room the following list may serve as a rough guide:

(1) Cranial nerves, excepting smell, taste, and vestibular tests. Acuity of vision not tested, except when patient indicates failing sight; only a rough orientation as to the visual fields, in the following way: the patient should face the observer and look constantly at a point between the observer's eyes. This one should hold his hands on either side of the patient's head, roughly in a frontal plane through the patient's eyes, and level with these. If the patient can see the fingers of both hands moving under these conditions, there can be no hemianopsia of the common types (homolateral and bitemporal).* *N.B.*—Ophthalmoscopic examination in all cases complaining of headache.

(2) Selected tests for motor system and co-ordination:

(*a*) Hand grip.
(*b*) Quick finger movements.
(*c*) Dorsiflexion at the wrist-joint.
(*d*) Diadochokinesis.
(*e*) Flexion at hip-joint.
(*f*) Walk on heels and tiptoe.
(*g*) Romberg's test, and in this position:
    (*a*) Finger-nose test.
    (*b*) Bending backwards, and
    (*c*) Standing on either leg.

(3) Rough orientation as to the patient's deep and superficial pain sensations. Finger-heel-pointing test Stereognostic sense.

* It has to be remembered, however, that some cases of bitemporal hemianopsia develop from more centrally seated bitemporal scotomata. In all cases suspect of pituitary tumour, therefore, a careful perimeter examination should be carried out.

(4) Reflexes: as in the preceding list.

(5) Rough impression of patient's mentality, judged by information gleaned during the examination.

[*N.B.*—Do not feel satisfied that a patient is necessarily sane, because in this rough way you find nil abnormal; and never give any written statement to the effect that the patient is of sound mind based on a single examination. (Apparently a fairly common mistake of practitioners in all countries!)]

This examination must, of course, also be completed in various ways, according to the nature of the case. The chief importance of such a standard routine examination is to ensure that in all cases the *whole* nervous system passes review.

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